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Pulmonary Hypertension*

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PULMONARY hypertension is not a disease. It occurs as a secondary manifestation of a large group of pathologic conditions, some of which are admittedly rare. The recognition of pulmonary hypertension is especially important for two reasons: 1. It frequently affords the crucial clue to the diagnosis of an otherwise obscure disease. 2. It affords a clearer concept of the disturbed physiologic condition present, thereby serving as a guide to a more rational therapeutic approach.

The reason that pulmonary hypertension has been relegated to relative obscurity arises out of the following facts: there is no practical method for measuring pulmonary pressure; there are no superficial pulmonary vessels to inspect or to palpate; there is nothing comparable to the ocular fundus for indicating the state of the arterioles of the pulmonary circulation, as the retinal arteries do in cases of essential hypertension. Pulmonary hypertension is recognized, therefore, by its secondary effects on the cardiorespiratory system which results in a syndrome to be described later, which usually can be recognized easily.

The diseases affecting the parenchyma of the lungs have overshadowed so completely the pathologic condition affecting its vascular elements, that we often lose sight of the fact that the pulmonary circulation is actually a part of the cardiovascular apparatus. Moreover, the similarity of symptoms resulting from primary heart disease to those dependent on pulmonary conditions readily tends toward confusion, unless the physiologic mechanism in each case is clearly understood.

The simple arrangement found among the lower vertebrates, such as fishes, serves the excellent purpose of reminding us of the functional and anatomic inseparability of the pulmonary and systemic vascular systems. The heart in these instances contains only venous blood and discharges its contents by a system of efferent vessels into and through the gills where gaseous exchanges occur. A corresponding group of efferent vessels conducts the oxygenated blood from the gills to the systemic circulation.

The human cardiorespiratory system, although it has evolved into a complicated mechanism anatomically, corresponds with the same plan functionally. With the development of a lung for respiratory functions, the venous heart in the form of a right auricle and right ventricle still serves the purpose of dispatching the unoxygenated blood by way of the pulmonary arterial tree into the respiratory system for gaseous exchanges. The efferent vessels, however, have acquired a pumping mechanism of their own in the form of the left auricle and a left ventricle. For the purpose of understanding the effects produced by diseases of the pulmonary vessels, it is important to visualize constantly this functional continuity and interrelationship of the venous and arterial hearts.

In discussing conditions affecting the lesser circulation, it must be remembered further that the pulmonary arterial tree, like the remainder of the vascular system, is endowed with a vascular area and a capillary bed greater than ordinary functional demands can exceed. The presence of structural changes in the pulmonary circuit, therefore, does not imply necessarily that there has been any limitation of function[1]. As long as the "reserve" is not encroached on to a serious extent, symptoms may be in abeyance and the cardiorespiratory apparatus may adapt itself to new conditions and remain compensated. Among experimental subjects, Haggart and Walker[2] have shown that 52 to 66 per cent of the pulmonary circulation could be cut off without significant variation in the general circulatory condition. The point at which failure occurs is sharply defined and beyond this point, circulatory collapse is precipitated by even a minute increase in the arterial obstruction. Underhill[3] sums up the position as follows: "The healthy heart, therefore, can accommodate itself without difficulty to sending the same volume of blood through one lung only in a given time, as it previously sent through both."

RESULTS AND EFFECTS OF PULMONARY HYPERTENSION

Any mechanism which leads to increased pressure in the pulmonary circuit, irrespective of its mode of production, increases the work of the right ventricle and, if sustained, as it usually is, will lead to hypertrophy of that chamber. As secondary effects on the pulmonary vessels themselves, there follow arteriosclerotic changes in the main pulmonary arteries and added obstructive effects in the arterioles. Over a period of time, when the reserves are exhausted, failure of the

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right side of the heart may ensue wth resultant dilatation, venous congestion and edema. When pulmonary hypertension is the result of obstruction within the pulmonary circuit, the interference with ventilation and the resulting anoxemia eventually exert deleterious effects on the nutritional status of the left side of the heart, and so set up a vicious cycle. As Waring and Black[4] point out, the collateral circulation which may be afforded by the bronchial arteries cannot in any way compensate for the respiratory difficulties inasmuch as these vessels carry oxygenated blood.

ETIOLOGIC FACTORS AND MANNER IN WHICH EACH CAUSES PULMONARY HYPERTENSION

The conditions resulting in increased pressure within the pulmonary circuit are varied, but they can be arranged conveniently into two groups, depending on the mechanism involved: (1) obstruction to the pulmonary circuit either beyond or within the pulmonary system and (2) arteriovenous shunts in certain cases of congenital heart disease.

OBSTRUCTION OF THE PULMONARY CIRCUIT

Obstruction beyond the pulmonary circuit. Mitral stenosis constitutes the classic example of a mechanical obstruction to the circulation beyond the pulmonary circuit. This results in increased pulmonary pressure. Oxygenation is not interfered with until compensation fails, because the obstruction is beyond the alveolar system. Enlargement of the left auricle eventually results.

A failing left ventricle, for any cause, be it systematic hypertension, aortic disease or myocardial degeneration, results in a similar train of events. With rise in pulmonary pressure, the right ventricle is compelled to contract against an increasing load. Dilatation of the left ventricle in the process of failure may be associated with two events which further embarrass the right side of the heart, namely, mitral insufficiency and bulging of the interventricular septum into the right ventricle, constricting its capacity and thereby interfering with emptying of the right auricle. Thompson and White[5] have shown that the effects on the right side of the heart resulting from left-sided strain alone may proceed to preponderant hypertrophy of the right ventricle and, in some cases, electrocardiographic examination gave evidence of right axis deviation. Accentuation of the second pulmonic sound in the course of hypertensive heart disease represents the clinical counterpart of such a state of affairs.

Obstruction within the pulmonary circuit. The obstruction may be at various levels of the pulmonary arterial tree.

I. The main pulmonary vessels.

A. Acute cor pulmonale. The sudden closure of a large pulmonary branch results in an equally sudden pulmonary hypertension, presumably because of arterial spasm in the associated pulmonary radicles[6]. If the accident is not fatal, the evidence of strain of the right side of the heart usually can be elicited clinically[7] and electrocardiographically[8,9].

B. Thrombosis of the pulmonary artery. A number of cases have been reported which presented clinically a picture of pulmonary hypertension and culminated in hypertrophy and failure of the right side of the heart. At necropsy, multiple thromboses, the origin of which was obscure, were found occluding many of the larger pulmonary branches. The reader is referred particularly to the reports of cases of Means and Mallory[10], Montgomery[11], Jump and Baumann[12], and Barnes and Yater[13] exemplifying this group. In the last named case, the history suggested that the arterial occlusions were of embolic origin.

II. Small and medium-sized pulmonary branches.

A. Sickle-cell anemia. The occurrence of cardiac enlargement, systolic murmurs and a palpable liver simulating rheumatic heart disease, have been noted frequently in association with sickle-cell anemia, when, at necropsy, evidence of valvular heart disease was not discoverable[14]. The assumption that these findings were the result of the anemia, of itself, has been questioned by Yater and Hansmann[15], who have reported illustrative cases in which the picture of failure of the right side of the heart was shown to have been caused either by thrombotic occlusion of the small and medium-sized arteries of the lungs or by thickening of the walls of the small and medium-sized arteries and the arterioles of the lungs with reduction in the size of their lumens, but without thrombotic occlusions. Capillary stasis owing to distortion and agglutination of erythrocytes was felt to be the essential factor in producing these arterial lesions.

B. Carcinomatous lymphangitis associated with obliterative pulmonary arteritis resulting in failure of the right side of the heart was described by Girode as early as 1889 according to Greenspan[13], who, in addition to reporting four such cases, gives an excellent and extensive review of the literature. The essential picture consists of widespread occlusion of arteries and arterioles apparently secondary to perivascular lymphatic infiltration with carcinomatous cells and proliferation of connective tissue incidental to this. Carcinomatous emboli occur within the vessels but play a relatively unimportant role in the obliterative process. By far the majority of cases result from a spread by way of the lymphatic vessels from a primary lesion in the stomach. The clinical course in such cases is much less protracted than in all other forms of chronic cor pulmonale and, thus, the term subacute cor pulmonale, as applied by Brill and Robertson[17], seems entirely justifiable.

C. Schistosomiasis (Mansoni) has been shown to cause granulomatous pulmonary lesions within or related to the blood vessels. On examination many small arteries gave evidence of extreme concentric thickening of the intima with diminution

of the size of the lumen and hypertrophy of the media in the case reported by Clark and Graef[18]. Right ventricular hypertrophy and congestive heart failure were present and there was no other ascertainable factor which might have caused pulmonary hypertension. It will be recalled that infection occurs through the skin and that the parasites are carried by the blood stream to the lungs, through which they are carried to be distributed by the systemic circulation, but ending mainly in the portal system. However, residual infestation in the lungs apparently can occur with secondary effects in the pulmonary vessels sufficiently widespread to cause pulmonary hypertension.

III. Pulmonary arterioles.

A. Secondary pulmonary arteriolar sclerosis occurs in association with a wide variety of conditions such as those already discussed, in certain cases of mitral stenosis[19], emphysema and pulmonary fibrosis, but these changes are not always sufficiently widespread to cause pulmonary hypertension in themselves.

B. Primary pulmonary arteriolar sclerosis is a rare and interesting entity in which no cause for these widespread arteriolar changes is ascertainable in either the pulmonary system or the cardiovascular system.

IV. Capillary system. When obstruction occurs to radicles of the pulmonary artery at levels proximal to the capillary bed, it is apparent that the corresponding sets of capillaries also are put out of commission automatically. Similarly, when the left side of the heart fails, capillary function is interfered with even if the capillaries are not altered structurally. Obliteration of portions of the capillary bed results further as a consequence of a group of pulmonary diseases, chief among which are emphysema (through destruction of the interalveolar walls) and widespread pulmonary fibrosis (which destroys portions of pulmonary tissue). Both these conditions, as a rule, are associated with chronic bronchitis; also asthmatic bronchitis and emphysema coexist in many instances. It is apparent that obliteration of the capillary bed must be much more widespread and extensive in order to exhaust the vascular reserves and to cause pulmonary hypertension than when the larger radicles of the pulmonary arterial tree are involved in such a process. This is an important point to grasp because it has a direct bearing on the controversy concerning the role of emphysema in the producton of pulmonary hypertension.

The association of emphysema and asthma with hypertrophy of the right side of the heart and congestive heart failure finds expression in the literature of the late Eighteenth Century and out of these observations the term "emphysema heart" was born. The historical background of our present conception of emphysema and its effects on the heart was adequately described by Parkinson and Hoyle[20] in a recent article. This problem cannot be pursued without incorporating two important observations in the discussion. The first is that advanced pulmonary emphysema may not be attended by hypertrophy of the right ventricle, increased venous pressure or other evidence of congestive heart failure and the circulation time through the pulmonary circuit maybe within normal limits even in the terminal stages of the disease[21]. There may actually be some venous engorgement associated with emphysema without any increase in venous pressure, however, this venous engorgement is the result mainly of increase in the intrapleural pressure which interferes with drainage of the venous blood into the right auricle. The second observation is that whenever pulmonary arteriolar obliterative changes are found, irrespective of the circumstances, hypertrophy of the right ventricle is present in a high percentage of cases with or without congestive failure[1].

It would seem that, if emphysema of the so-called obstructive type, which is a relatively frequent disease, were obstructive in the sense that it impedes the pulmonary circulation, there should be a far closer correlation between the degree and duration of emphysema on the one hand and evidence that the right ventricle has worked against resistance on the other. Most of the literature relative to the subject has failed to take into consideration the part which vascular changes (apart from capillary destruction) may have played in these cases in which hypertrophy of the right ventricle was found, accompanied by congestive failure. One ventures to predict that when representative surveys of cases of emphysema are completed, results will show that emphysema, of itself, does not cause pulmonary hypertension for, although the obliteration of capillaries is extensive in cases of advanced emphysema, it is extremely unlikely that it ever reaches a degree capable of exhausting the vascular reserve. In addition, evidence of failure of the right side of the heart, when it occurs, is usually owing to a second factor. This factor is either pulmonary arteriolar sclerosis, left ventricular failure for any cause, or valvular heart disease (such as mitral disease) in itself not developed sufficiently to have produced symptoms earlier. Also, it must be recalled that emphysema most frequently occurs among individuals who have reached the decades when hypertensive disease and disease of the coronary arteries are frequent.

It is of more than academic interest to determine whether pulmonary hypertension is present in cases of emphysema and pulmonary fibrosis or not, because it influences the prognosis very materially, and, in borderline cases, estimation of venous pressure may be necessary to decide this point. Patients who have uncomplicated emphysema, although they are forced to live a life of extreme limitation of activity, often give evidence of very little progression over several years but as soon as evidence of pulmonary hypertension can be elicited, the course is likely to be progressively downhill, eventually with uncontrollable congestive failure.

Kyphoscoliosis. Kyphoscoliosis presents another interesting problem. The observation that the majority of "hunchbacks" ultimately succumb to heart failure often has been made[22]. In 154 cases studied by Bachmann[23], 87 gave evidence of hypertrophy of the right ventricle, 27, of the left ventricle and 40, of both ventricles. Here we are probably dealing with several factors rather than with any single mechanism. Thus, large portions of lung often are atelectatic and are associated with other regions of compensatory emphysema; chronic bronchitis is frequent and the mechanical effect of kinking of the great vessels unquestionably plays a part in producing a picture which tends to terminate in failure of the right side of the heart.

ARTERIOVENOUS SHUNTS IN CASES OF CONGENITAL CARDIAC ANOMALIES. The anatomic arrangement in certain cases of congenital heart disease is such that the pulmonary circulation receives an added load of blood, through shunts from the arterial side and in this manner establishes increased pressure in the pulmonary circuit.

Patency of the ductus arteriosus. Because the pressure in the aorta is considerably higher than in the pulmonary artery, an extra quota of blood escapes through this channel into the pulmonary artery with each cardiac cycle. The usual evidences of pulmonary hypertension soon become manifest.

Septal defects. In most cases of septal defects the reflux of blood from the arterial to the venous side is not enough to cause any circulatory embarrassment unless a secondary factor supervenes which increases the arteriovenous shunt. A classic example of this type is so-called Lutembacher's disease consisting of a combination of mitral stenosis and a patulous foramen ovale. In such cases extreme degrees of pulmonary hypertension with dilatation of the pulmonary artery to aneurysmal proportions may result.

Transposition of the great vessels. The transposition of the arterial trunks usually is associated with an incomplete interventricular septum and varying degrees of pulmonic stenosis, resulting in right ventricular hypertrophy. This syndrome, the tetralogy of Fallot, in reality, is an example of chronic cor pulmonale but right ventricular hypertrophy is dependent more on pulmonic stenosis than on an admixture of arterial and venous blood.

DISCUSSION OF THE CLINICAL PICTURE OF CHRONIC COR PULMONALE WITH SPECIAL REFERENCE TO DIFFERENTIAL DIAGNOSIS

In short, the clinical picture of chronic cor pulmonale is that of pulmonary hypertension plus the features contributed by the factor responsible for the pulmonary hypertension. In cases of so-called primary pulmonary arteriolar sclerosis, the distinct syndrome of pulmonary hypertension occurs without the presence of any such discoverable factor to obscure or to distort the clinical picture. There is usually a history of gradually increasing dyspnea as the obliterative process progresses. The resulting interference with ventilation gives rise to cyanosis which also increases as the disease advances. The striking symptom, then, is dyspnea which is prominent even in the absence of congestive heart failure and is out of all proportion to other evidence of congestive heart failure, when the right side of the heart does fail. In short, it is the dyspnea of interference with ventilation and this in itself immediately should suggest its pulmonary origin. It is a characteristic of all pulmonary diseases in which the function of oxygenation is limited.

By the time these patients present themselves for examination, the secondary manifestations of pulmonary hypertension usually can be elicited. The individual often looks older than his chronologic age would imply and this is accentuated by the cyanosis which is present. The striking auscultatory finding is an accentuated second pulmonic sound. The clinical evidence of a hypertrophic right ventricle is afforded by a systolic pulsation to the left of the sternum best elicited by firm palpation in this region. This is accounted for by the fact that, as the right ventricle becomes hypertrophic, it comes to form, more and more, the anterior surface of the heart and approximates the anterior wall of the thorax more closely. On auscultation, the lungs are likely to be clear even in the presence of congestive failure, because the obstruction is proximal to the alevolar system. It is only when the left side of the heart fails secondary to the effects of anoxemia that congestion of the bases of the lungs comes into the clinical picture.

Confirmatory evidence is afforded by laboratory procedures. Roentgenologic examination reveals a prominent pulmonary conus, an enlarged right ventricle and auricle and unusually clear pulmonary fields. Pulsation of the vessels in the pulmonary hilus sometimes can be elicited. An important distinguishing feature is the absence of enlargement of the left auricle which distinguishes it from obstructions beyond the pulmonary circuit as in the cases of mital stenosis. Finally, the characteristic electrocardiographic pattern indicative of predominant right ventricular strain affords corroborative evidence. This consists of right axis deviation which is usually definite and if changes in the T waves occur, they will consist of inversion in the second, third and fourth (standard) leads. There may be secondary polycythemia, the result of anoxemia.

With failure of the right side of the heart, venous congestion, hepatic enlargement, peripheral edema, ascites and increasing cyanosis are added to the picture and it is characteristic of this disease that, when congestive failure supervenes, the subsequent course is invariably rapid downhill, a type of congestive failure unusually refractory to therapy.

This description holds essentially for pulmonary hypertension owing to any cause, the provoking

disease, either in the pulmonary system or cardiovascular apparatus, adding its quota to the symptoms and findings described. Pulmonary hypertension secondary to diseases of the left side of the heart and to the congenital lesions enumerated needs no special comment because the corresponding clinical picture in each case bears hallmarks sufficiently well recognized to establish a diagnosis.

The ability to distinguish between the various processes which cause pulmonary hypertension by obstructing various levels of the pulmonary arterial tree depends on (1) a knowledge of the possible pathologic conditions which are capable of producing such obliterative vascular disease, (2) an understanding of the mechanism involved in each case, that is, the life history of the disease and (3) a deliberate attempt to elicit evidence of pulmonary hypertension when diseases are encountered which are known to be capable of produsing pulmonary hypertension. Because some of these diseases are likely to obscure the characteristic findings of pulmonary hypertension, valuable prognostic data may be overlooked unless a special effort is made to search for such evidence.

The term Ayerza's disease purposely has been omitted from the discussion because this name has been linked with so many varied pathologic and clinical entities that it contributes neither etiologic nor differential diagnostic information.

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Lesions of the Lips and Oral Cavity

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MIDWAY between medicine and dentistry there is a field belonging partly to both professions, neglected by the medical man. and usually beyond the training and knowledge of the dentist. That field comprises the conditions affecting the lips and the lining of the oral cavity. Some of these lesions are the oral manifestations of cutaneous diseases; others are peculiar to this location while still others are a part of the picture of various systemic diseases. The mouth offers a wealth of clues which are useful in the diagnosis of disease elsewhere, and routine examination of the mouth yields a surprising amount of pathology.

It is not our purpose to give a detailed or complete account of the diseases which may affect the lips and oral cavity, but rather to mention briefly those lesions which in our experience are most likely to be met with and are of most practical importance to the physician.

Such conditions as pyorrhoea, which principally concern the dentist, have no place in this discussion. The oral lesions of the acute exanthemata are also purposely omitted, as are the rarer neoplasms.

It is our hope that this presentation will stimulate interest in the routine examination of the oral cavity as part of the general examination, and will aid in the interpretation of the findings.

Certain skin diseases will show at times lesions of the oral mucosa. Lichen planus, lupus erythematosus, pemphigus and erythema multiforme are especially likely to develop mouth lesions. Their

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character is more or less changed by their environment. Their presence is often sufficient to confirm a diagnosis of atypical skin disease.

Such serious diseases as syphilis, pellagra, pernicious anemia, agranulocytosis, pemphigus, and Addison's disease are often first suspected on account of typical or suggestive changes in the oral mucosa. In about half the cases of pernicious anemia Moeller's glossitis is the earliest recognizable sign of the disease.

Patients not infrequently come to the dermatologist or radiologist with a cancer-phobia caused by the misinterpretation of some benign or trivial lesion of the tongue or oral mucous membrane. On the other hand, search of the mouth and lips for pre-malignant lesions will often reveal these when the patient is not aware of their presence.

The mouth, particularly the gums, may give a clue to metallic intoxications and overtreatment with metals. The bluish gray of argyria, the black or near black line of bismuth, the bluish black of lead, and the bluish gray of mercury are well known.

Addison's disease shows a fairly constant brownish discoloration of the oral mucosa.

The characteristics of the various lesions to be considered can best be given briefly in tabular form:

I LESIONS PECULIAR TO THE LIPS

LESIONS	DESCRIPTION	ETIOLOGY
Cheilitis Actinica	Dryness, scaling, fissuring, hyperkeratosis	Sunshine
Cheilitis Venenata	Erythema, scaling, vesiculation	Contact irritants
Cheilitis Exfoliativa	Persistent desquamation	Unknown
Cheilitis Glandularis	Prominent mucous orifices, swelling, moisture	Congenital defect
Perleche	Maceration and scaling at commissures	Monilia. Streptococcus
Herpes Simplex Labialis	Grouped vesicles	Filtrable virus

II LESIONS PECULIAR TO THE TONGUE

LESIONS	DESCRIPTION	ETIOLOGY
Scrotal Tongue	Furrowed, swollen	Syphilis, congenital defect
Black Hairy Tongue	Black or dark brown filaments	Tobacco, chemicals, congenital defect, fungus
Geographic Tongue	Chronic, changing, superficial, desquamated plaques	Unknown. Possibly indicates some neuropathic anomaly
Burning Tongue (Glossodynia)	Burning pain without visible lesion	Pellagra, pernicious anemia, neurosis, burn from dissimilar metals
Glossitis Rhombica Mediana	Chronic, rhomboidal or oval red plaque, on dorsum	Developmental defect?
Moeller's Glossitis	Red, superficial areas, burning and painful	Pernicious anemia. Pellagra
Syphilitic Sclerosis (Interstitial Glossitis)	Pink or whitish, smooth areas on dorsum	Tertiary syphilis

III LESIONS WHICH MAY OCCUR ON LIPS, TONGUE AND ORAL CAVITY

LESIONS	DESCRIPTION	ETIOLOGY
Apthae	Small grouped ulcers	Filtrable virus
Periadenitis mucosa necrotica recurrens	Recurrent infiltrated ulcers	Occurs in anemic women
Vincent's Angina	Acute superficial ulcerations	Spirillum and fusiform bacillus
Agranulocytic Angina	Acute ulcerations and stomatitis	Agranulocytosis
Thrush	White patches	Monilia
Pemphigus	Bullae, red, denuded areas	Obscure. Infectious?
Erythema Multiforme	Red, denuded areas	Toxic
Lichen Planus	Smooth white flat papules	Neurogenic?
Lupus Erythematosus	Light, atrophic patches	Toxic
Angioneurotic Edema	Acute swelling	Allergic
Pellagrous Stomatitis	Red, denuded areas	Vitamin deficiency
Stomatitis Medicamentosa	Redness, desquamation	Drugs (toxic effect)
Stomatitis Venenata		Contact irritants (drugs, dentrifices, foods)
Electric Burns	Redness, swelling, pain, metallic taste	Dissimilar metals in mouth
Fordyce's Disease	Small yellow spots in mucosa	Anolamous sebaceous glands
Leucoplakia	White plaques	Tobacco, syphilis, condiments

IV LESIONS WHICH MAY OCCUR ON LIPS, TONGUE AND ORAL CAVITY (continued)

LESIONS	DESCRIPTION	ETIOLOGY
Tuberculosis	Deep fissures, painful undermined ulcers	Usually secondary to pulmonary or laryngeal tuberculosis
Syphilis—Chancre	Indurated ulcer	Primary syphilis
Syphilis—Mucous Patch	White or denuded patch	Secondary syphilis
Syphilis—Gumma	Painless, indolent nodule or deep ulcer	Tertiary syphilis
Pyogenic Granuloma	Rapidly growing, highly vascular tumor	Staphylococcus
Mucous Retention Cyst	Shiny, translucent tumor	Occluded mucous duct
Hemangioma	Blood vascular tumor	Congenital
Lymphangioma	Pseudo-vesicular appearance	Congenital
Fibroma	Firm tumor	Congenital
Lipoma	Soft, lobulated tumor	Congenital
Papilloma	Flat or pedunculated tumor	Infectious or traumatic
Epulis—Fibroma Fibroma—angioma Giant cell	Tumor on gum. Arises from periosteum	Infection, trauma, poor hygiene
Epithelioma	Growing, infiltrating tumor	Tobacco, rough teeth, infection
Sarcoma	Firm, growing tumor	Unknown

CONCLUSION

The lesions likely to be found on the lips and in the oral cavity have been presented in tabular form, along with a very brief description, and the etiology in those cases in which it is known or suspected. We hope that these tables will serve as a reference for the busy practitioner.

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Congenital Umbilical Hernia

With Report of Strangulated Case

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CONGENITAL umbilical hernia, also called exomphalos, omphalocele and hernia funiculi umbilicalis must not be confused with the comparatively common acquired umbilical hernia of infants.

Congenital umbilical hernia is a questionable term since in these cases the organs involved have probably never been in the abdominal cavity, but developed extra-abdominally, as noted in this paper. The condition is very rare. Available statistics indicate its occurrence in 1:6000-1:10,000 deliveries. Of this number a much smaller percentage contains the liver. In an extensive seacrh of foreign and American literature we found only one previous case reported of a strangulated congenital umbilical hernia involving the liver and gall bladder, as was true in our case. Here again the condition is not to be confused with an eventration or fissure of the anterior abdominal wall. The only opening into the peritoneal cavity must be through the umbilicus, which in our case measured about 1½ cms. in diameter.

Historically we find that Ambroise Pare described this condition in 1634 and there were nine cases published prior to 1800. In 1809 Antonio Scarpa of Italy, differentiated between congenital umbilical hernia and acquired type and reported two cases, one containing a lobe of the liver.

The literature up to 1900 contains less than 200 cases reported from the entire world. Altpeper reported 112 cases in 1930 for the period of 1900-1929 in the world literature. From 1929-1937 there were 46 cases reported.

It is obvious that a great many cases were not reported. Various reasons account for this:

1. Small and unrecognized.
2. Associated with other gross anomalies wth early death.
3. Still births.
4. Mid-wife deliveries.

ETIOLOGY

The etiology is not clear. These are developmental anomalies just as club-feet, spina bifida, etc., but with no hereditary tendency. They occur most often in males. Their origin is traced to the development of the abdominal wall. It must be remembered that in the embryo a portion of the foregut develops in the exocelom, normally being forced into the peritoneal cavity about the third month; also all the peritoneal contents are exposed directly to the exocelom. Closure of the cavity is effected by growth of the amnion and amniotic somatopleure. The incomplete extension of the amnion and failure to completely surround the viscera results in their further development in the exocelom and are found at birth as hernia into the cord, usually with a thin peritoneal covering. Thus we see how it is possible for any tissue of the peritoneal cavity to be found in such hernia but obviously such tissues as the liver, spleen and urinary bladder are found only seldom. The liver and gall bladder arise as buds from the foregut, while the

latter is still outside the body cavity. They should enter the cavity about the 7th week.

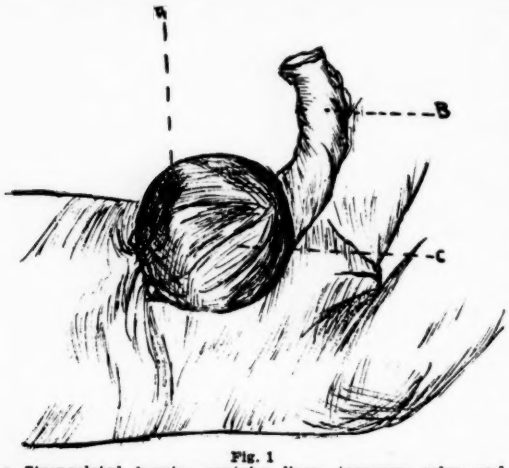

Fig. 1
a. Strangulated hernia:—contains liver; tranverse colon and loops of small intestine.
b. Umbilical cord.
c. Coverings { cord covering / Peritoneum.

DIAGNOSIS

The diagnosis is rather easy when seen early the cord tissues are fairly transparent and the contents can be seen. The hernia appears as a variable sized mass presenting as picture dependent on its contents. The liver or spleen present a dark firm mass of considerable size. A loop of the bowel or piece of omentum will be much smaller and peristalsis may be noted. The mass may be reducible. Impulse on crying may be felt. After a few hours Wharton's jelly becomes opaque and offers more difficulty in diagnosis, although it is still easy if the possibilities are remembered.

TREATMENT

The treatment is surgical. Obviously since the rare nature of the condition precludes anyone claiming to be an authority from experience (A. F. Herbert of New Orleans had three cases, the greatest number reported in American literature) we must gain our opinion from the reports of others. Here we find that in all cases listed the mortality with expectant treatment is three times that of operative, namely about 70%. The surgical mortality is about 23% in a large series. These include mostly cases of intestinal contents. Of five cases containing the liver only one lived. Of these, one was reported late and the liver was not replaced in the abdomen and in my own case gangrenous changes had occurred due to strangulation.

The inevitable outcome of expectant treatment appears to be a drying of Wharton's jelly as is always seen in post-partum cords, with subsequent rupture of the sac, and probable peritonitis. It is true that in very small, reducible type treated with alcohol packs an occasional cure results, but the chances are against it.

Surgery should be done as early as possible because small infants tolerate surgery fairly well, and the intestinal content is probably sterile up to eleven or twelve hours. Strangulation due to shrinking of the cord is less apt to occur. There is a very definite danger in cases left more than a few hours.

Great care should be taken to avoid shock. It is well to place the child on a heating pad during surgery unless the room is very warm. Infection should be avoided by careful preparation of the abdomen first with olive oil, then ether, soap and alcohol.

The operaton should be done as rapidly as possible. The incision will probably have to be made downward in cases involving the liver to avoid injuring the falciform ligaments and entering the pleural cavity through the diaphragm which is probably drawn down with the liver. This was particularly noted in our case. No attempt is made to close the abdomen in layers, through and through sutures are used. This is advocated by most operators. No attempt is made at fixation of the liver or other organs.

The post-operative care consists in maintaining body heat; supplying fluids by 25 c.c. injections hypodermically of normal saline or saline with 2½ to 5 per cent glucose every four to 5 per cent glucose every four to six hours. Water or gelatin water with dextrose by mouth and breast milk as early as possible should be given.

REPORT OF CASE

Baby V., delivered by a mid-wife at 5:45 P. M., was seen by Dr. Jumper at 7:00 P. M. because the parents were worried about the tumor. Dr. Chas. Rennick, pediatrician, was called in consultation and I was then called as surgeon in the case.

We found a four-hour-old infant with a mass 7x5x4 cms. covered by the cord structure and along the inferior margin the cord vessels were seen with some strands of the mid-wife's hair tied with the ligature. Wharton's jelly had lost its transparency. The mass was firm, dark and smooth; no pulsation was noted on crying and no peristalsis. The Child had been vomiting since birth. Temperature was 98.4; pulse rate 146 and respiration 32. The blood findings were: white blood cells, 34,000; red blood cells, 6,900,000; Hemoglobin, 108%; coagulation time, 2½ m. No other fetal malformations were noted.

Immediate surgery was done with no anesthesia. The child was quiet for the most part and relaxation was adequate. The liver was carefully freed from the adherent cord structures. The gall bladder presented in its normal location with its ducts. The ligaments and lobes of the liver appeared normal in size but the entire mass, including the transverse colon and several loops of small intestine had suffered considerably from strangulation.

The incision was carried nearly to the symphysis to permit replacement of the liver. The margins of the abdominal opening were trimmed and the wound closed by through and through sutures. The child was placed in an incubator. Fluids were given.

The following day it did well; but the third day it died. Evidently the strangulation and surgery were too much for it. However, I believe that had this case been delivered by a physician and immediate surgery done, the child might have lived.

COMMENT

The question of anesthesia in such cases is open to question. We have all seen circumcisions per-

formed without anesthesia and have probably been told that a young infant does not feel pain. I wrote to Dr. C. B. Courville, professor of Neuroanatomy of College Medical Evangelists, Los Angeles, and he replied: "I have never had occasion to look into the matter which you refer to in your letter. I see no reason why an infant should not be endowed with sensation since the sensory fibers and tracts are myelinated at the time the baby is born. To be sure, the sensory association areas are not well developed and there may be some difficulty in the painful sensations, so that when the child has a sensation it might not fully interpret it as a disagreeable one. This seems a bit fanciful though, for I see no reason why this should be present only for a few days after birth and then the normal situation arise as far as painful stimuli are concerned."

I believe the explanation lies in the failure to interpret pain sensation as such. This is surely feasible since a new-born infant evidently has not learned to differentiate the pleasant from unpleasant sensations.

Certainly I have seen numerous infants circumcised without any apparent distress other than their crying due to restraint, and if given a nursing bottle they would apparently be unmindful of cutting and suturing. In our case the child was quiet during most of the procedure.

Anesthesia was unnecessary in so far as relaxation was concerned, since the musculature is so delicate as not to interfere. Difficulty in placing the viscera in the peritoneal cavity is not due to muscle spasm but to crowding into an unaccustomed place.

For those strict anatomists and physiologists who question the absence of sensation because the tracts are there we would ask: "What about a hypnotized subject?" The tracts are there. No chemical or physical block is present. Yet the mind fails to react to outside stimuli except at the will of the hypnotist.

The question is worthy of study.

SUMMARY

1. Congenital umbilical hernias differentiated from acquired.
2. Rare. 1:5- 10,000.
3. May contain any abdominal organ.
4. Covered by peritoneum and Wharton's jelly usually.
5. Early surgery essential.
6. Delay means rupture of sac, strangulation or both.
7. Mortality in all cases about 22%.
8. Mortality in cases with liver much higher.
9. No anesthesia needed.

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The Intravenous Use of Fluids*

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ONE of the significant advances in modern medicine has been the development of satisfactory and safe intravenous methods of supplying fluids, foods and chemicals to patients whose condition is such that they cannot or should not take sufficient fluid by mouth, or whose condition may be distinctively benefited by the administration of certain specific types of fluid. Whether or not this has been an unmixed blessing should be a subject for contemplation. Certainly, as far as the comfort of the patient is concerned, the advantages of the intravenous administration of fluids over hypodermoclysis cannot be doubted. That the patient has gained comfort but has sacrificed safety owing to the injudicious use of such a dangerous method by the unskilled cannot be denied. Although the skilled use of solutions intravenously in proper amounts and composition is frequently a lifesaving measure, the improper use of such solutions certainly may do the patient harm. In the last few years, since such solutions have been properly prepared and the "reactions" following their injection have been largely eliminated, this method of administering fluids has gained wide popularity. The amount of fluid necessary, the type of fluid to be used and the rate of injecton all have been the subject of much controversy and expermentation. Cutter, from a study of the output of a commercial laboratory making fluids for intravenous use, found that

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5 per cent d-glucose in physiologic saline solution was the most widely used, this type of mixture comprising 36 per cent of the solutions distributed for intravenous use; 10 per cent d-glucose in physiologic saline solution was less widely used, and physiologic saline solution was the least widely used. The most interesting figures presented, however, were those which dealt with fluids customarily employed intravenously in various sections of the country. For example, in the Northwest, 62 per cent of the solutions used were 5 per cent d-glucose in physiologic saline solution, whereas, in the Southwest this solution was used in only 14 per cent of cases. Such a variation can only mean a wide difference in the understanding of the physiologic principles involved in the use of intravenous fluids.

Before considering the various indications for the use of intravenous fluids, the type and amount of fluid best suited in each case, I should like to consider the matter of intake and output of fluids, that is, water balance in general.

Under normal conditions very little thought is needed for the maintenance of the fine adjustment which exists between intake and output of fluids. Nature has endowed the body with an excellent mechanism, the sensation of thirst, which goes into play when the supply of fluid needs replenishing. In answer to the often asked question as to how much water a normal person should drink each day, the answer is that it depends largely on the desire for water.

In any consideration of intake of fluid, naturally, we think first of the amount of water drunk and, frequently, the fact that half of the fluid acquired by the body comes from food is forgotten. The water in food is derived from two sources: (1) the content of water in the food itself which, if it consists of a normal, daily maintenance diet, will contain 1000 to 1500 c.c. of water and (2) water formed as the result of the oxidation of protein, fat and carbohydrate.

Loss of water from the body occurs mainly in three ways: (1) it is excreted in the urine; (2) it is excreted as the result of the regulation of the temperature of the body by sweating and evaporation and by the exhaling of water vapor and (3) water is lost in the stools. Under normal conditions the kidney can function with varying amounts of water, using what remains after other demands have been supplied. A satisfactory supply of water for renal function is nidicated by the formation of a dilute, that is, a light colored urine; on the other hand, an insufficient supply of water is indicated by the formation of a concentrated, that is, a highly colored urine. The second way in which fluid is lost is related to the control of the heat of the body through vaporization. Large amounts of fluid may escape practically unnoticed through the skin and breath. Newburgh and his associates have shown that healthy individuals carrying on activities such as the routine work of a nurse, a medical student or a technician will vaporize, under comfortable circumstances and without obvious sweating, 1000 to 1500 c.c. of water daily. As activity of the body and environmental temperatures increase, resulting in perspiration, the loss may increase in amount to several liters. In contrast to the water available for renal function which diminishes or ceases when the available supply is low, the heat regulatory mechanism of the body, by the process of vaporization utilizes what water is necessary and, if no other fluid becomes available, it will use more and more of body fluids until death results. The third way in which fluids are lost from the body, mainly through the feces, normally accounts for not more than 150 c.c. daily and, therefore, is negligible.

WATER BALANCE IN ABNORMAL STATES

There are many conditions in which parenteral and, especially, intravenous administration of fluids becomes necessary. Among the more frequent conditions are: (1) in postoperative conditions, (2) in cases of diabetic coma and other types of coma, (3) in cases of renal disease, (4) cases in which nausea and vomiting are present, (5) cases of intestinal obstruction and (6) conditions of shock and hemorrhage Among those conditions which may be benefited by the intravenous administration of fluids mainly on account of the type and concentration of certain constituents of the fluid rather than on account of the fluid itself are: (1) disease of the coronary arteries, including myocardial infarction, (2) cirrhosis of the liver and (3) certain injuries of the head. In the treatment of each of these conditions there are certain specific indications regarding the amount and type of fluid to be used and the consideration of certain factors will be helpful in the choice of fluids. The factors are: (1) amount of fluid necessary for physiologic function; (2) dehydration; (3) physiologic considerations of the blood plasma; (4) renal function and (5) "extrarenal" factors.

REQUIREMENTS FOR FLUID

Coller and Maddock[3] have shown that the amount of fluid required by the surgical patient who has an uncomplicated, postoperative course is as follows: (1) water for vaporization, 1500 c.c. and (2) water for the formation of urine, 1000 c.c. If there is an abnormal loss of fluid, such as that caused by vomiting, the administration of additional fluid to compensate for this loss is necessary. For the patient who is suffering from sepsis or who has fever, additional fluid will be required, as follows: (1) water for vaporization, 2000 c.c.; (2) water for the formation of urine, 1500 c.c. and (3) additional fluids to replace abnormal losses, a total of not less than 3500 c.c. It is obvious that when patients, in hot weather, lose large quantities of fluids owing to perspiration or when perspiration is excessive following high fever and chills, or when there is diarrhea, several additional liters of fluid may be required to keep a balance between intake and output of fluids. The magnitude of abnormal losses of fluid owing to vomiting, loss of blood and drainage from biliary and intestinal fistulas is frequently not realized even when there is evidence of a lack of

water balance, such as the presence of oliguria with anuria.

DEHYDRATION

On the day of operation, a certain amount of dehydration takes place among the majority of patients. The intake of food and fluid is restricted and, in addition, there are losses of fluid from the body. The operating room is warm, the patient is carefully wrapped and perspires profusely following operation. This is frequent observation. Furthermore the loss of fluid as a result of loss of blood may be considerable. Pilcher and Sheard at The Mayo Clinic studied the loss of blood by patients on whom transurethral prostatic resection had been performed. They found that in 33 per cent of an initial series of fifty-five patients the loss of blood was 600 c.c. and the average loss of blood for the whole series was 479 c.c. After an improvement in the resectoscope had been made, a second series of fifty-five patients was studied; only 15 per cent of this group had a loss of blood of more than 600 c.c., and the average loss of blood was 291 c.c. Maddock reported the results of a study of loss of fluid in a group of eighteen patients undergoing general surgical operations. The total loss of fluid owing to loss of blood, vomiting, formation of urine and vaporization amounted to an average of 1 liter. Following operation, it is customary to wrap the patient in blankets for hours, which procedure results in further perspiration, and there may be vomiting. As a result, a patient is usually moderately or severely dehydrated postoperatively.

Dehydration occurs very frequently among men who have urinary obstruction owing to prostatic enlargement. Frequently, these patients find by experience that the more fluid they drink, the more trouble they have and, as a result, oftentimes enter the hospital severely dehydrated, as is shown by sunken eyes, drycoated tongue, uremic breath and dry, inelastic skin. Another group of patients who come to us in a severely dehydrated condition are those who, owing to vomiting secondary to intestnal obstruction or other disease, have not only been unable to take and retain fluids but have drawn on the reserves of fluid in the tissues through continuous loss of gastric and intestinal contents. Not only has vomiting resulted in a large loss of fluid, but there has been a serious loss of chloride and acid which must be considered when fluids are to be replaced. Although the Wangensteen apparatus for applying continuous duodenal suction is one of the most valuable methods of treating intestinal obstruction, it must be remembered that its use continuously depletes the body of fluids and electrolytes. The amount of fluid necessary to establish an adequate water balance in the presence of dehydration may be as much as 7 liters. Coller and Maddock[5] have calculated this as follows: water for vaporization, 2000 c.c.; water for the formation of urine, 1500 c.c. and fluid for hydration, 3600 c.c. They calculated this amount from the results of studies on two healthy adult subjects. They found that the loss of fluid which was sufficient to cause the usual clinical signs of dehydration amounted to about 6 per cent of the body weight. Therefore, this amount must be replaced before a proper balance can be established.

Seven liters seem to be a large amount of fluid to administer to a patient, but we have frequently observed that this amount is necessary before an adequate urinary output can be obtained. Root and Riseman have recently reported excellent results in the treatment of prolonged diabetic coma by the intravenous injection of as much as 11,000 c.c. of normal saline solution and large quantities of insulin in the course of a few hours.

RELATION OF DEHYDRATION TO RENAL FUNCTION

The maintenance of a normal water balance is so intimately associated with renal function that the effects of an insufficient amount of water available for purposes of excretion and the effects of dehydration on the kidneys excrete 35 to 40 gm. of solids met and Newburgh have shown that, under normal conditions, the kidneys excrete 35 to 40 gm. of solids in twenty-four hours. Each gram of this waste material requires 15 c.c. of fluid to be dissolved. Therefore, even with kidneys working at their maximal capacity for concentration, at least 500 to 600 c.c. of urine must be excreted in twenty-four hours to prevent the retention of nitrogenous waste products in the blood. If renal damage is present and if the kidneys cannot concentrate to a maximal specific gravity of 1.032, more fluid than 600 c.c. is required to eliminate the total of solids. The amount of fluid required for the elimination of 35 gm. of waste material will increase with the decreased ability of the kidneys to form a concentrated urine. These data are contained in table 1. That renal injury may be the result of prolonged dehydration is indicated by the presence of albumin, casts and erythrocytes in the urine.

With dehydration there occurs hemoconcentration which results in an increased viscosity of the blood. This, probably hinders the circulation through the kidney. Medes and Herrick have shown that decreased blood flow through the kidneys leads to a diminution of glomerular filtrate. Dehydration also increases the concentration of total blood proteins which, by increasing the colloidal osmotic pressure of the blood, leads to diminished renal function. Thus, when, at times, the urinary output becomes diminished or ceases and it appears that the kidneys "have shut down," our first thought should be whether or not sufficient fluid is available so that the kidneys can function.

PHYSIOLOGIC SOLUTION OF SODIUM CHLORIDE

Thus far I have considered mainly the amount of fluids which should be administered when parenteral fluids are needed. The kind of fluid is also of extreme importance. In order that the kidneys may function at maximal efficiency, the blood plasma must be as nearly normal as possible. The guiding principle in the decision of what type of

fluid should be used should be to supply what the patient needs.

The routine intravenous administration of solutions of sodium chloride, or solutions of sodium chloride in combination with d-glucose, without much thought being given to their physiologic effects seems to be a frequent practice. The normal daily intake of salt is between 6 and 10 gm. Physiologic saline solutions contain 8.5 gm. of sodium chloride. Thus, if 4000 c.c. of such a solution were administered in twenty-four hours, the patient would receive 34 gm. of salt. Certainly, if fluids were administered by mouth no one would think of asking the patient to consume that amount of sodium chloride. Furthermore, whereas normal kidneys have a remarkable ability to excrete sodium chloride in excess of what is necessary to maintain a normal concentration in the blood plasma, it is likely that the prolonged administration of excessive amounts of salt may result in renal damage or, at least, may favor the retention of excessive amounts of salt in the tissues. This results in edema owing to the retention of fluid. The concentration of sodium chloride in the blood plasma has no direct relation to the amount of edema present. In fact, the content of chlorides in the plasma may sometimes be less than normal in the presence of edema. Wakefield and Keith have reported the deleterious effects of intravenous administration of solutions of sodium chloride in cases of chronic glomerular nephritis.

The development of edema postoperatively among patients whose convalescence has been slow, who have suffered from prolonged fever and who have had daily injections of physiologic saline solution is a matter of frequent observation. In the care of the average surgical patient, the intravenous injection of saline solution is carried out for not more than two or three days, until adequate fluid can be taken by mouth so that the development of edema is rarely observed. If edema is present, it will not appear in the legs where early edema is usually encountered in the case of ambulatory patients, but will be seen, in the dependent portions of the body, by turning the patient so that the back may be inspected. With the resumption of taking fluids by mouth, the edema that has developed promptly disappears. The amount of edema is usually directly related to the length of time the patient receives the intravenous injections of saline solution. Coller, Dick and Maddock have recently called attention to this fact. They found that twelve of the thirteen surgical patients who received 5 per cent d-glucose in physiologic saline solution or in Ringer's solution gained weight as a result of retained fluids. Administration of Ringer's solution was slightly less likely to cause retention of fluid than was that of physiologic saline solution. On the other hand, fluid was not retained by members of a group of surgical patients who were given 5 per cent d-glucose in distilled water. Furthermore, in those cases in which edema developed as the result of the excessive administration of solutions of sodium chloride, the use of solutions of d-glucose was shown to be very effective in the elimination of retained fluids. Jones and Eaton and, more recently, Curphey and Orr have called attention to the occurrence of edema among surgical patients owing to another factor, namely, the lowering of the concentration of blood proteins incident to inanition. Gilligian and his associates have recently shown that inanition may not be the only factor in the reduction of the concentration of plasma proteins, but that the concentration of plasma protein also decreases as the result of the intravenous administration of fluids. The former authors call attention to the importance of restricting the intake of sodium chloride at the first sign of edema. As a result of these observations, they stress the value of repeated transfusions of blood when adequate protein cannot be given by mouth.

Whether or not the development of a small amount of edema is harmful to the patient is not definitely known. It is more than likely that the presence of mild edema for short periods is not harmful. Severe and prolonged edema is a serious complication which should be prevented, if possible, and should be treated vigorously if present. Edema interferes with cardiac function, reduces its efficiency and favors the development of pulmonary edema. Edema definitely diminishes the emptying time of the stomach, whether through the pylorus or through a gastro-enteric stoma. This is an important postoperative consideration. Usually, the sicker the patient, the more fluid he is given, and the finding of waterlogged tissues at necropsy would certainly indicate that such treatment had not contributed to the best interests of the patient. Recent experimental evidence would seem to indicate that the sodium and chloride ions have different functions and that the sodium ion may be more related to the formation of edema than the chloride ion.

On the other hand, it must be emphasized that insufficient chlorides in the blood plasma resulting from the loss of hydrochloric acid and fluid following prolonged vomiting may be equally serious. Under these circumstances, lowering of the concentration of plasma chlorides results in decreased renal function and the loss of the acid radicals results in alkalosis. Studies of renal function reveal an increased content of urea in the blood, a diminished output of phenalsulphonphthalein, and the presence of albuminuria and cylindruria. Inability to cope with this situation frequently leads to progressive toxemia, renal insufficiency and death. Microscopic study of the kidneys reveals, mainly, tubular degeneration. The treatment is the use of intravenous saline solution, thereby eliminating the etiologic factor.

When dealing with dehydration, the use of saline solutions seems beneficial for, as Gamble has pointed out, salt solution tends to be retained because it contains the substance required for reestablishing the volume of fluid.

SOLUTIONS OF D-GLUCOSE FOR INTRAVENOUS USE

In the absence of specific indications for the ad-

ministration of sodium chloride, a solution of 5 or 10 per cent d-glucose in distilled water seems to be ideal for intravenous use. When little or no food is taken by mouth, the glycogen content of the body is rapidly diminished, and fat, which then becomes the main source of energy, is incompletely oxidized, with a resultant ketosis caused by the formation of beta-oxybutyric and aceto-acetic acids. D-glucose also provides a considerable source of energy; for example, 1500 c.c. of 10 per cent d-glucose provides 600 calories. It must be remembered, however, that metabolic balance cannot be maintained by d-glucose, sodium chloride or water intravenously, so that patients must have food by mouth at the earliest possible time.

Solutions with concentrations of d-glucose greater than 5 or 10 per cent should be used only when specifically indicated. Solutions of 20 or 25 per cent or even 50 per cent d-glucose are frequently effective in the mobilization of edema or in the presence of anuria or oliguria and for other conditions which will be mentioned.

SOLUTIONS FOR REGULATION OF THE ACID-BASE EQUILIBRIUM

A discussion of the mechanisms of the body for the regulation of acid-base equilibrium is not within the scope of this discussion. Alkalosis may occur following the prolonged use of alkaline powders in the treatment of peptic ulcer. This is frequently seen in cases of renal damage. Acidosis occurs most frequently as the result of (1) renal insufficiency and (2) neglected diabetes. The following report of a case illustrates many of the problems encountered in the management of a patient who had acidosis secondary to renal insufficiency with urinary obstruction secondary to prostatic enlargement.

A man, aged 84 years, had experienced symptoms of increasing urinary obstruction for three years. Finally, he was entirely unable to void and it was necessary that the bladder be catheterized. He had been vomiting frequently. On admission to the hospital, January 5, 1937, he was extremely dehydrated, very weak and stuporous. The prostate gland was enlarged, grade 4 on the basis of 1 to 4, the enlargement being caused by benign hyperplasia. He was given 2000 c.c. of physiologic saline solution intravenously immediately on admission after an inlying urethral catheter had been inserted (table 2). Physiologic saline solution was used in this case for it was assumed that because the patient had been vomiting, the concentration of blood chloride might be reduced. This was subsequently found not to be the case. The elevated concentration of blood chlorides in this instance may have been related to the factor of dehydration. The use of physiologic saline solutions intravenously in cases of dehydration may have a beneficial effect in aiding the body to retain fluids until a satisfactory water balance has been established. A study of the carbon-dioxide combining power of the blood showed the presence of definite acidosis. This was combatted by the use of 500 c.c. of a 5 per cent solution of sodium bicarbonate intravenously. As the acidosis was moderately severe, it was considered that a second injection would be necessary. This was carried out before the results of the studies of blood chemistry were known; thus, alkalosis resulted as was indicated by the fact that the carbon-dioxide combining power was 80.6 volumes per 100 c.c. of plasma.

Although this patient did not suffer ill effects and improved daily, the case is cited to illustrate the necessity for caution and the importance of frequent chemical studies of the blood when sodium bicarbonate is used intravenously. One injection of 500 c.c. of a 5 per cent solution of sodium bicarbonate, in this instance, would have been sufficient to combat the acidosis. In general, not more than one injection of the solution should be given without studying the carbon-dioxide combining power of the blood.

DEXTROSE SOLUTIONS IN HEPATIC DISEASE

The value of a high carbohydrate, low-fat and low-protein diet and the intravenous injection of solutions of d-glucose are well known adjuncts in the preoperative and postoperative management of patients suffering from disease of the biliary tract. Obstructive jaundice is followed rapidly by progressive injury to the hepatic cells, thus interfering with storage and manufacture of glycogen. The same is true in cases of cirrhosis of the liver. Important clinical data have been derived from the study of experimentally produced lesions of the liver among animals. Bollman and Mann, by ligating the common bile ducts of animals, were able to show many of the phenomena which are associated with obstructive jaundice in man. During the first few days following ligation of the duct, the liver becomes enlarged, but soon becomes smaller than normal and there is extensive atrophy of the hepatic cells. As time goes on, it becomes increasingly necessary to give a high carbohydrate diet to keep the animals alive. After two or three months, if a diet consisting exclusively of meat is given, the animals will die, but if a diet rich in carbohydrates is given, life may be prolonged for at least a year.

Bollman and Mann have been able also to produce experimentally a picture similar to the usual clinical signs of portal cirrhosis by the repeated administration of carbon tetrachloride. The administration of alcohol alone did not produce necrosis of the liver but the administration of alcohol and a diet rich in fats produced changes which were rapidly lethal. The addition of alcohol to hepatic toxin produced much more extensive lesions than when hepatic toxin was given alone. The administration of alcohol produces fatty changes in the liver which reduce hepatic resistance and, therefore, the liver is unable to combat other poisons effectively. More extensive lesions are produced when the animals are maintained on a diet of meat than when they are maintained on a diet high in content of carbohydrate. Toxic agents are much more effective in the production of cirrhotic changes in the liver of the fasting animal than in the liver of an animal which has been given food rich in carbohydrate. These studies seemed to show definitely that the administration of solutions of d-glucose is of definite value following surgical procedures on animals whose livers are extensively injured, that the intravenous injection of solutions of d-glucose in large amounts seemed to have brought about the recovery of many animals that otherwise almost certainly would have died. The recuperative power of the liver was also demonstrated even when there was evidence of advanced cirrhosis. Among animals at least, it is not a hopeless condition if the toxic agent can be removed and, if the animal can be maintained on a high carbohydrate diet, the symptoms of ascites and jaundice disappear and the general condition of the animal improves. Jones at the Massachusetts General Hospital and others

have stressed the value of a high intake of d-glucose for patients who have jaundice. The intravenous administration of solutions of d-glucose has always seemed to be associated with a greater content of glycogen in the liver than when carbohydrate is taken by mouth. However, the recent work of Althausen and Stockholm seems to refute this hypothesis. They have shown that in mice at least the administration of d-glucose by mouth will cause as great a concentration of glycogen in the liver as when it is given intravenously.

The serious outlook for patients who have advanced portal cirrhosis is well known. It was during the time that Bollman and Mann were conducting their experiments on the production of cirrhosis in animals that a patient showing all the evidences of advanced cirrhosis presented himself at the clinic for treatment. The results of the use of d-glucose intravenously in this case seem worthy of reporting.

A jeweler[21], aged 58 years, who used alcohol in moderation, registered at the clinic in August, 1935, complaining of jaundice, swelling of the abdomen and lower extremities and weakness. These symptoms had been noticed for about six weeks. On examination, the patient was weak, jaundiced, orthopneic and slightly cyanotic. The abdomen was greatly distended with fluid and there were numerous distended, superficial veins over the abdominal wall. Edema of the legs and ankles, grade 2 (on the basis of 1 to 4), was present. The patient was immediately hospitalized for treatment and for completion of his examination. Urinalysis revealed the presence of a trace of albumin, occasional hyaline and granular casts and bile. The values for blood urea and blood sugar were within normal limits; that of serum bilirubin fluctuated considerably, the average level being 5 to 6 mg. per 100 c.c. of serum. Roentgenologic examination of the thorax and electrocardiographic studies gave essentially negative results; the result of the Takata-Ara test was positive. The amount of benzoic acid excreted in four hours was 1.5 gm. representing an approximate reduction of 50 per cent.

Treatment directed toward the relief of ascites proved to be of little avail, there being practically no response to diuretics. On the second day after admission, it was necessary to perform paracentesis and about 3 liters of straw-colored fluid was withdrawn. After this procedure was accomplished, a very firm liver could be felt, the edge of the liver was palpable about a hand's breadth below the costal margin. The spleen was not palpable. Fourteen days after admission, still improvement was not noted and a further paracentesis was necessary. At this time, 9 liters of slightly blood-tinged fluid was recovered. Because the patient failed to respond to treatmetnt and because of the rapid recurrence of ascites plus mental symptoms, it was felt that the prognosis was extremely poor and it was decided to permit the patient to return to his home. We concluded that it would be well to disregard the edema and ascites and continue with daily injections of solutions of d-glucose intravenously in conjunction with giving a high carbohydrate diet, in the hope that some hepatic regeneration might be induced.

The patient's brother-in-law, a physician, undertook the daily intravenous administration of d-glucose, 100 to 300 gm. in solution. A month after dismissal from the clinic, the doctor reported slight improvement with regard to the edema and ascites, but stated that the mental symptoms had not improved, the patient being very restless and confused most of the time. The daily administration of solutions of d-glucose intravenously, however, was continued and an occasional injection of liver was administered intramuscularly. It had been necessary to perform paracentesis about once every two or three weeks and 10 liters of yellowish fluid was removed on each occasion. At this time, persistent glycosuria developed which required the administration of 10 to 20 units of insulin daily for its control.

In November, the patient's physician reported that improvement had been slight but that abdominal paracentesis was necessary only every three weeks. The patient still lapsed into a confused mental state at intervals, these episodes alternating with periods of restlessness. In January, 1936, five months after the daily intravenous injections of d-glucose had begun, definite improvement was apparent. The ascites had practically disappeared, the urine was free of sugar and evidence of jaundice could not be found. In March, 1936, the patient's physical condition had returned practically to normal. In September, 1936, one year after treatment had been begun, the patient returned to the clinic for re-examination. He was able to care for his business and do light work about his summer cottage. Examination at this time revealed the presence of a slight icteric tint of the sclera. The edge of the liver was palpable at the costal margin. Anemia was not present and the urine was normal. Roentgenologic examination did not reveal esophageal varices. The concentration of bilirubin was 4.4 mg. per 100 c.c. of serum and, as might be expected, there was some retention of dye on testing hepatic function. The frequency of intravenous injections of solutions of d-glucose had been gradually reduced as the patient improved. Frequent reports from the patient and his physician have indicated that the patient is in excellent physical condition: symptoms of disease are not present and he is able to carry on with his usual duties. Our last letter from the patient is dated January 21, 1938. In this letter, he stated: "I have never felt better in my life. I had pneumonia in November, was ill about four weeks but was on the job for Christmas business."

SOLUTIONS OF D-GLUCOSE FOR INTRAVENOUS USE IN CORONARY SCLEROSIS

The use of solutions of d-glucose intravenously in varying concentrations has been a most valuable aid in the management of patients suffering from myocardial damage secondary to coronary sclerosis and in cases of myocardial infarction. Dyspnea, especially the paroxysmal, nocturnal type is, in many instances, greatly relieved by the daily injection of 300 to 500 cc. of a 10 or 20 per cent solution of d-glucose intravenously. As in cases of hepatic disease, the use of d-glucose solutions intravenously seems to replenish the content of glycogen of heart muscle more effectively than does d-glucose by mouth and this probably accounts for its beneficial effects. When edema is present, the amounts injected should be somewhat smaller than otherwise, but owing to the diuretic effect of concentrated solutions of d-glucose the mobilzation of edema may be aided.

SOLUTIONS FOR INTRAVENOUS USE IN MISCELLANEOUS CONDITIONS

In the presence of shock, the use of solutions intravenously is extremely valuable and their prompt use is frequently a lifesaving measure. In this condition, treatment is aimed at raising the blood pressure and restoring the volume of circulating blood. Solutions of physiologic saline or d-glucose may be used but, inasmuch as these solutions leave the circulation rapidly, they are not as effective as solutions of acacia or a transfusion of blood. The amount of solution of acacia usually given is 450 c.c.; the solution used consists of 6 per cent acacia in a 0.9 per cent solution of sodium chloride. The giving of a solution of acacia intravenously is not as effective as a transfusion of blood but it may be used in the treatment of milder states of shock or as a temporary measure until blood can be obtained.

The use of concentrated solutions of d-glucose (20 to 50 per cent) intravenously may be helpful in certain cases of internal hemorrhage, to combat pulmonary edema and increased intracranial pressure (unless cerebral hemorrhage is present). The value of giving solutions of saline and d-glucose intravenously in the treatment of Addison's disease is well known.

In the presence of oliguria or anuria, the use of hypertonic salt solution in the form of 10 per cent sodium chloride may be very effective in reestablishing a satisfactory urinary output. Similarly, 20 per cent, 25 per cent, or even 50 per cent solutions of d-glucose in distilled water may be effective in producing diuresis.

PRACTICAL CONSIDERATIONS IN USING FLUIDS INTRAVENOUSLY

The rate of injection of fluids intravenously is a

problem which demands consideration. The cardiovascular system, under normal circumstances, compensates very readily for the additional load placed on it by the intravenous injection of fluids. The fluids diffuse into the tissues or are eliminated rapidly so that an increase in the volume of blood is only slight and temporary. Usually 50 to 75 drops per minute can be well tolerated. Sometimes, in the presence of myocardial damage, the intravenous injection of fluids will cause some substernal pain or a sensation of pressure in the chest and dyspnea. The rate of injection should then be reduced below the point at which symptoms are produced or the injection should be discontinued. Sometimes, one or two hours are necessary for the injection of 1 liter of fluid.

Solutions of d-glucose injected intravenously especially those of high concentration, will cause venous thrombosis, an important consideration when frequent injections are necessary over a long period of time. This may be obviated, in many instances, by administering, after the injection of the solution of d-glucose, a few cubic centimeters of a physiologic saline solution. The amount of solution safe for injection at one time, at various ages, has been tabulated by Lundy, Osterberg and Tuohy (table 3). They give as the maximal amount 1000 c.c. for one injection. However, at times, we have given as much as 2000 c.c. without noting untoward results.

Sometimes venipuncture is difficult on account of small veins. This difficulty may be overcome to a great extent by covering the arm, wrist and hand with a hot, moist pack for thirty minutes before the injection is made. When intravenous treatment is to be carried out for several days, the veins used may be marked by color or may be charted so that the same vein will not be used too frequently.

SUMMARY AND CONCLUSIONS

1. The use of solutions intravenously is a valuable aid, frequently lifesaving, but, if not properly carried out both as to the type and quantity of fluid, harm may result.

2. The normal water balance of the body is maintained without conscious effort. Fluids are derived from those drunk, from food and from the oxidation of food. Fluids are lost in the urine, by vaporization, and in the stool.

3. It is frequently necessary to administer fluids parenterally (1) after surgical operations, (2) in cases of diabetic coma and other types of coma, (3) in renal disease, (4) nausea and vomiting, (5) in intestinal obstruction and (6) in shock and hemorrhage.

4. The amount of fluid required under average conditions and in the presence of dehydration is discussed. The latter condition may require as much as seven to ten injections daily before a fluid balance is established.

5. The excessive use of solutions of physiologic saline intravenously may be followed by the development of edema. When loss of chloride has occurred from vomiting and excessive perspiration, sodium chloride must be replaced.

6. The use of alkaline solutions intravenously to maintain acid-base balance is discussed and an illustrative case is cited.

7. Solutions of d-glucose (5 to 10 per cent) in distilled water may be used intravenously when sodium chloride is not specifically needed.

8. The value of solutions of d-glucose intravenously in cases of hepatic and cardiac disease is discussed. A case is reported showing the benefi-

TABLE 1

Minimum amount of water required to excrete 35 gm. waste material

Status of kidney	Maximum concentrating ability of the kidney: Specific gravity	Water needed, c.c.
Normal	1.032—1.029	473
Disease	1.028—1.025	595
	1.024—1.020	605
	1.017—1.015	850
	1.014—1.010	1439

TABLE 2

Blood findings and intravenous medication, in a case of urinary obstruction, showing effect of administering sodium bicarbonate intravenously

(Case 1)

January	6	7	8	9	10	11	12	13	14	15
Urea (mg./100 c.c.)	174		90				48			48
Creatinine (mg./100 c.c.)	2.2		1.5							
Chloride (mg./100 c.c.)	651		600							558
Carbon dioxide combining power (vols./100 c.c.)	21.1		57.9	80.6			66.4			55.1
Intravenous medication										
Physiologic saline solution (c.c.)	2000							2000	2000	1000
5 per cent glucose in distilled water (c.c.)		1000	1000	1000	1000	1000	1000			1000
5 per cent sodium bicarbonate (c.c.)		500	500							

TABLE 3

For the average person: safe dosage of various substances for intravenous administration, in c.c.

Substance	Age of patient: 3 months and younger	6 months	1 year	2 years	5 years	10 years	15 years	20 years and older	Remarks
Blood	75	100	150	200	250	300	400	500	May repeat after 12 hours
Acacia 6%									
NaCl 0.9% (phys. NaCl)	100	150	200	300	350	500	750	1000	
D-glucose 5% in phys. NaCl									
D-glucose 10% in phys. NaCl									
D-glucose 5% in aq. dest.									
D-glucose 10% in aq. dest.									
Hartman's solution									
D-glucose 20% in aq. dest.	75	100	150	200	250	300	400	500	Give very slowly
Sucrose 25%									
$NaHCO_3$ 5%									
D-glucose 50% in aq. dest.			5	10	25	50	75	100	To reduce intracranial pressure

cial results of prolonged intravenous injection of solutions of d-glucose in a case of advanced portal cirrhosis.

9. Some practical considerations in the use of fluids intravenously are discussed.

Mayo Clinic.

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DISCUSSION

Dr. L. B. Cohenour (Albuquerque) opening:

"I am sure we have all enjoyed Dr. Habein's fine paper, which I will not attempt to discuss, other than to say that the greatest offender in the use of fluids intravenously is the irregular practitioner who uses everything under the sun."

Dr. S. L. Burton (Albuquerque):

"I want to say that I appreciate Dr. Habein's paper. I am not too old to learn and I have gained much from this paper."

Dr. Habein (closing))

"It is quite likely that we have used too much intravenous saline solution. Some years ago when glucose was first used, reactions consisting of chills and fever were quite common. This was undoubtedly due to the impurities in the solution and probably bore a direct relationship to the reactions following the use of intravenous glucose are no more tions are now properly prepared and reactions following the use of intravenous glucose is no more frequent than with the use of saline, intravenous glucose becomes an ideal solution. It must be remembered to have a normally functioning kidney, there must be a normal blood plasma. If the chlorides in the blood are low, saline must be given. On the other hand, if they are normal there is no reason for the use of intravenous saline solution. As soon as patients are able to take fluids by mouth, the amount of intravenous solution should be gradually reduced.

Fractures of the Spine With Injuries to the Cord

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THE subject of fractures of the spine with injuries to the cord is so vast that I will stress only a few of the points which may not be generally understood, and are consequently neglected in treating patients with these injuries.

Fractures of the spine are of great importance from the moment the injury occurs, not alone because of the effect on the bony structure and its function, but also because of the spinal cord being contained within the canal. Unfortunatetly these fractures are frequently unrecognized, and the patient at first is treated for a strained back. Even the less serious cases, when not properly treated, will cause persistent pain resulting in possible deformity, and at times, permanent disability. An extensive bone injury, however, may occur with complete recovery, if the spinal cord is not involved, provided the patient has received proper treatment.

Fractures of the spine can be classed under three groups: compression fractures of the bodies; fracture-dislocation; and fractures of the articular facets, the spinous and transverse processes, and the laminae. Compression fractures occur in more than half of the fractures of the spine; they result from a forced hyperflexion with the crushing of the body of the vertebra into a wedge or saucer shape. The loose cancellous bone fragments may spread in any direction; if this spread be backward, the cord will be compressed. Fracture-dislocations of the spine are by far the most serious of these groups as the vast majority are accompanied by spinal cord lesions. Understanding the anatomy of the spine, one can readily see that in any severe injury to the bony structure, fracture without dislocation is exceedingly rare, that injury to the articular facets, the intervertebral discs, and the neural arch are common with an occasional tearing of the ligaments. The most frequent causes of this are the crushing of the flexed spine by heavy objects falling upon the head, back or shoulders, by automobile

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accidents in which one is thrown from the car onto the ground. Fractures of the spinous and transverse processes and laminae are rare; they are usually due to direct violence, more rarely to muscular action. If both laminae are fractured and displaced, spinal cord damage is likely to occur.

Fractures of the spine are increasing in frequency, and with the ever increasing speed and power of the automobile, more of these injuries will occur annually. These fractures result in high percentage of temporary and permanent disaiblities as well as fatalities. The seriousness of these injuries is often aggravated by lack of proper emergency care. Simple linear fractures are frequently converted into displaced or fracture-dislocations with damage to the cord through improper handling. Well-meaning but inexperienced presons attempting to give first aid to the injured person at the site of the accident or attempting to transport him to the hospital may often be as dangerous to the patient as the accident itself. Far too frequently we see patients with fractures of the spine arriving at the hospital in a cramped or sitting position, having been lifted at the site of the accident with no support to the spine, placed in the first available automobile, and rushed over roads and streets at a high rate of speed. That every jolt and turn should damage the spine, and possibly the cord, seems inevitable.

FIRST AID

When any accident victim complains of pain in his neck or back, there is the chance of a fracture of the spine. Never move such a person until he has demonstrated mobility in the upper and lower extremities. If he is unable to move the upper extremities, he may have a fracture of the cervical vertebra; if he is unable to move the lower extremities, he may have a fracture of the dorsal or lumbar vertebra, and an injury to the spinal cord. Though immediate recognition of a fracture is most important, one should not attempt to prove the diagnosis at the site of the injury. If the victim is unconscious, then by all means he should be handled as if he had a fracture of the spine. Accordingly the first consideration in the treatment of fractures of the spine is the immediate, careful handling of the injured person.

When the patient is first found at the site of the accident, he is usually in shock and pain. Therefore, he should be immediately covered with a blanket to conserve body heat; if necessary, he should be given morphine to combat shock and to relieve pain. Keep the patient on his back, or on his abdomen, at the exact spot where he is found until experienced help and a proper supporting splint can be obtained. The splint must be taken to the patient wherever he may be. Do not move him until the splint is at hand.

A padded "ladder" splint or a rigid stretcher should be used to support the patient for transportation to the hospital. A ladder splint consists of two long boards fastened together by three or four cross pieces nailed or tied. Placing the injured person upon the ladder splint must be done by three or four persons who understand what they are to do. They must first lay a blanket by his side and gently roll him upon the blanket so that he rests on his abdomen, arms by his sides, with his head turned. He may then be lifted in the blanket and placed on the ladder splint, or a rigid stretcher.

If a fracture of the cervical vertebra is suspected, one person should exert light traction on the head; another, on the feet, while the other two lift beneath the shoulders, trunk, and hips. They must keep his head, body, and lower limbs horizontal and place him on the ladder splint on his back. Thereafter he should be tied to the splint or stretcher to prevent any movement and to keep him in a prone position while being transported to the hospital. Upon arriving at the hospital, the patient should be kept in this position until examination and definitive treatment can be undertaken. If there is severe shock, this condition must be treated first; that can be done, of course, without any change of position. First save the patient, you can treat the fracture or other injuries later.

EXAMINATION AND DIAGNOSIS

If, on the other hand, the patient's condition will permit, make a careful physical examination; many patients with fracture of the spine have other injuries also requiring attention. The Soto-Hall sign is an aid to diagnosis. With the patient flat on his back, the examiner places one hand upon the sternum, exerting a slight pressure so no flexion can take place either at the dorsal or lumbar regions. At the same time the examiner's other hand is placed under the occiput, and with this hand the head is bent upon the neck; then slowly but forcibly, the head and neck are flexed upon the sternum. This produces a progressive pull upon the posterior spinous ligaments, starting at the ligamentum nuchae and being transmitted downward to the entraspinous ligaments until the spinous process of the injured vertebra have been reached. Acting as a lever gently compressing the body, this produces pain which the patient can localize very accurately.

Anteroposterior and lateral roentgenograms of the whole spine should be taken; the latter of these is the more important. Oblique views are frequently required to show the fracture. A negative finding is not positive proof, however, that a fracture does not exist. There are compression fractures of the vertebral bodies which may not be apparent immediately after the injury, but which will be demonstrated some days later. Collapse of fractured vertebra has occurred after negative x-ray findings. One should not overlook a fracture of the spine because neither symptoms of cord injury nor deformity are found upon physical or x-ray examination. Delayed bone and cord symptoms often develop unexpectedly. Be on the lookout for late manifestations.

SPINAL CORD INJURY

Injuries to the spinal cord as a result of fracture of the vertebra present a difficult problem. The im-

mediate damage to the cord at the site of the fracture may include laceration, hemorrhage, or edema, or all three together. Laceration causes permanent damage to the cord substance with degeneration of the ascendng and descending tracts. Hemorrhage is usually most extensive in the central gray matter. Edema occurs soon after the injury, and extends over a number of segments above and below the level of the injury. Edema of the substance of the cord may produce a cord compression, which, if not relieved in six hours, will result in a permanent cord damage with secondary softening and loss of function. Contusion of the cord with edema may occur in spinal injuries in which no fracture can be demonstrated. Occasionally displacement of the vertebra occurs with a crushing or severance of the cord; thereafter the vertebra snaps back into normal position, leaving no x-ray evidence to account for the cord lesion. Cord damage may result from elongation by angulation of the vertebra, from a partial or complete compression, from a partial or complete separation of the segments of the cord, or from bony fragments penetrating the cord.

The immediate signs of spinal cord injury are a partial or complete interruption of conduction with impaired, or lost motion and sensation below the site of the injury. With symptoms of cord damage, there is always uncertainty regarding the exact pathological condition of the cord. A partial or complete paralysis of muscles below the cord lesion may be due to pressure from adjacent bone, to edema of the cord, to hemorrhage into the substance of the cord, to a combination of any of these factors. The x-ray is of great assistance in determining the injury to the bony structure; it may show whether or not the neural canal has been narrowed by the impingement of the surrounding bone; but unfortunately, as mentioned before, the extent of the injury to the body canal is not a certain guide to the damage done to the cord. A neurological examination will reveal partial or complete lesion with stationary, increasing, or diminishing symptoms. In case of cord damage a Queckenstedt test should be performed to determine the degree of spinal cord injury, to prove whether a partial, complete, or no block exists.

Regeneration of the non-neurilematous elements of the spinal cord will not occur, consequently, following a compression of the cord by edema, hemorrhage, or fracture for more than six hours, or a division of the cord between the atlas and the first lumbar vertebra, no improvement may be expected, except through extra spinal nerve anastamosis. Nerve injury below the first lumbar vertebra is capable of regeneration when it is relieved of pressure over a much longer period.

When complete paralysis occurs immediately, the prognosis is extremely grave; there may be either a severe compression or a severance of the cord. Accordingly recovery is not to be expected. If the paralysis is gradual in onset, it is probably due to edema or hemorrhage; if partial, one should suspect there is a partial compression of the cord or a partial severance by fragments of bone in the cord.

TREATMENT

As soon as diagnosis of fracture of the spine and its effect upon the cord is made, provided the patient's condition will permit—and it must be remembered that even manipulation is poorly tolerated by these cases—definitive treatment should be instituted. Restoration of vertebral contours and normal alignment are essential to complete restoration of the vertebral structure and the spinal cord.

I will not attempt to describe the methods of closed reduction by traction, manipulation, and hyperextension. These and the standard methods of immobilization are undoubtedly familiar. A review of the literature seems to show that avertin is the anesthetic of choice in these cases. Personally, I prefer evipal or pentothal sodium because of their quick induction of anesthesia, and their controllability with a rapid return to consciousness. Infiltration of a one per cent solution of novocain into the site of the fracture frequently gives a satisfactory anesthesia. Certainly we should know within a short time after an attempt at closed reduction if our objective has been obtained. That quick return to consciousness is a very definite help in determining the state of the spinal cord after attempted closed reduction.

When there is a fracture of the spine with deformity and a paralysis, neither of which has been relieved by a diligent attempt at closed reduction, prompt determination of the future course of action is necessary. Remember, when a cord is compressed beyond six hours, you have a permanent injury. The longer the cord compression persists, the less are the chances for recovery. Again the Queckenstedt test should be used to aid in the diagnosis.

A case of fracture of the spine with abolition of motor and sensory function below the level of the fracture which has not been improved after a diligent effort of closed reduction, can usually be classed as a complete transverse section of the cord, although complete physiologic block of the spinal cord may exist without an anatomic block being present. We have no test to aid us in a determination as to whether there is an anatomic or physiologic block. Even the splendid Queckenstedt test fails in showing this.

The surgeon should be neither a conservative nor a radical; he must be guided by circumstances. Laminectomy in these cases is to be considered very seriously. While laminectomy is attended by a high mortality, yet if it is to be beneficial, it must be done within six hours after the injury. Even though recovery may occur without laminectomy, serious results frequently follow failure to relieve a remediable condition. Quite often laminectomy offers the only possible hope for improvement or recovery. Although only a few may be cured, still quite a number can be restored to fair health and usefulness. These patients are exceedingly grateful and happy as a result of the operation. I feel that, in case of doubt, the patient should be given a chance for recovery whenever beneficial results may follow the operation.

There are certain definite indications for laminectomy: i.e., after closed reduction when improvement occurs for a while, but later ceases short of satisfactory cord function; when the paralysis develops several hours or a day or two after the accident, making quite certain the symptoms are due to cord edema or hemorrhage; when the paralysis is only partial, or did not occur instantly after the accident, but came on gradually; when bone encroachment on the spinal canal or a spicula of bone in the canal is shown by roentgenograms; when nerve injury or compression below the first lumbar vertebra occurs.

If the articular facets, the intervertebral discs, the spinal ligaments, and the vertebra have been injured by the accident, they may be further injured and weakened by the laminectomy; therefore, when a laminectomy is performed, protection of the spinal structure and the cord against further damage and deformity is of greatest importance.

On November 26, 1913, I had a patient who sustained a severe fracture-dislocation of the twelfth dorsal and first lumbar vertebra with angulation of the spine and loss of motion and sensation below the site of the injury. An attempted closed reduction failed to correct the deformity or relieve the paralysis. She was then prepared for operation and anesthetized. A four-inch incision was made with the central portion over the injured vertebra. Free hemorrhage had occurred and the supra and infraspinous ligaments between the eleventh and twelfth dorsal and first and second lumbar vertebra were lacerated. Both lamina of the twelfth dorsal were completely fractured and displaced inward, pressing upon the cord, so all that was necessary to expose the cord was to dissect the soft tissues from the spinous process and lamina and lift the bony fragment from its bed. The right lamina of the first lumbar vertebra was fractured. Removal of the bony fragment of the twelfth dorsal revealed a cord compressed by angulation at the junction of the twelfth dorsal and first lumbar vertebra. The cord compression was relieved by cutting through the left lamina of the first lumbar and removing the bony fragment. The cord soon regained its normal fullness and appeared uninjured. The membranes were punctured with a needle and clear cerebrospinal fluid withdrawn. The wound was closed and dressed. Motor and sensody functions had returned when the patient recovered from the anesthetic.

Because of the damage to the vertebra I decided to do a bone transplant to strengthen the spinal column and protect the cord against damage. Three weeks after the primary operation, the transplant operation was performed after the technique of Dr. Fred H. Albee. This was the first reported bone transplantation as a treatment for fracture-dislocation of the spine, given by me before the 23rd annual meeting of this association and in this city on April 22nd, 1914. This patient made a perfect recovery, and has had an active normal life since, and has been in the best of health.

A considerable number of other cases properly selected have done equally as well. A strong bony transplant for an internal splint gives immediate immobilization, gives immediate support, and extension of the spine with protection to the spinal cord. Bony union between the transplant and the spinous processes above and below the injury provides the spine with a firm supporting power almost up to the normal. It interferes very little with the usual spine mobility. With a loss of the lamina and the spinous processes of two or more vertebra, the bone transplant is of distinct advantage. As a rule the bone transplant should not be done at the time of the laminectomy. The patient is usually in too serious a condition. It is better to do it as a secondary procedure two weeks or longer after the laminectomy.

SPINAL FUSION

Though spinal fusion is of advantage in some cases of fracture of the spine, it should not be done immediatetly. If after conservative treatment pain and disability continue, then fusion should be considered seriously and done if it offers hopes of improvement. A plaster shell should be prepared before either of these operations is performed; the patient is immediately placed in the shell to obtain fixation. He should remain there for six or eight weeks; after that a properly fitting brace should be worn for three to six months. The length of time that any support for fracture of the spine must be worn will depend upon the physical and x-ray findings. Proper treatment should return about sixty-five per cent of these patients to their pre-injury activities in from six to twelve months.

CONCLUSIONS

Fractures of the spine should be suspected when a victim of an accident complains of pain in the neck or back. If unconscious, he should be handled as if he had a fracture of the spine.

The speed of application, the type of first aid, the proper supporting splint for transporting, and finally careful transportation are major factors in the ultimate recovery of the patient with a fracture of the spine.

An early accurate physical and x-ray diagnosis, together with an immediate restoration of vertebral contours and normal alignment, are essential for complete restoration of function of the vertebral structure and the spinal cord.

Injuries to the spinal cord present a difficult problem which must receive immediate careful consideration.

Compression of the spinal cord for more than six hours will result in a permanent damage to the cord as regeneration of the non-neurilematous elements of the cord does not occur.

Use closed reductions whenever possible, and resort to laminectomy whenever it offers the only possible hope for improvement or recovery.

Both bone transplantation and spinal fusion have a definite place in the treatment of fractures of the spine; they should be used when indicated.

11 East Monroe.

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Lead Absorption and Lead Poisoning

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HISTORY OF LEAD POISONING

THE ancients as far back as 370 B. C. recognized lead as the cause of symptoms. Hippocrates described a severe attack of colic in a man that extraced metals. Nicander, in the second century B. C., understood the relationship of constipation, abdominal pain and pallor to the action of lead in the body. Dioscorides, in the first century B. C., said that the drinking of litharge (red lead) caused oppression of the stomach, belly and intestines with intense wringing pains. It suppresses the urine while the body swells and acquires an unsightly leaden hue. It causes paralysis and delirium.

In 1000 A. D., Avicenna recommended purgatives for lead colic. In 1656 Stockhusen recognized lead poisoning following absorption of lead dust. Tronchin, in 1757, pointed out the similarity of symptoms occurring in individuals drinking lead-sweetened wine and in lead workers. In 1767 Sir George Baker traced the lead colic to the lead employed in the manufacture and storage of cider. In the beginning of the 19th century Grisolle described colic, lead line and encephalopathy very accurately. A few years later Tanquerel des Planches published his book on lead poisoning and very little has been added to our clinical knowledge of the disease since that time, with the exception of the treatise on lead palsies written by Dejerine-Klumpke in 1889.

The first record of experimental work on lead poisoning was done in 1814 by Orfila, who administered lead intravenously and by mouth. Since that time and up to a few years ago nothing of interest was discovered until Aub and his co-workers, through their research, gave us our present day knowledge of lead poisoning as concerns the mode of entry, pathology, mode of excretion and treatment.

LEAD POISONING

Lead intoxication is the most common type of poisoning found in industry. However, we have cases of lead poisoning on record due to non-industrial sources such as water supplies, cosmetics, drugs and home-made beverages.

In the main, with a very few exceptions, industrial poisoning is due to the inhalation of lead, while non-industrial lead poisoning is due to the ingestion of lead.

There are many interesting records of non-industrial lead poisoning. In a study of 102 lead conducted water supplies in New England, 24% of 253 persons were leaded. It was estimated that the daily ingestion of 0.1 mgs. over a period of 8 years caused this leading. The United States Public Health Bureau has the ruling that water containing one-half part lead per million parts of water should not be used for drinking purposes. Lead poisoning from water supplies has been reported from England, France, Germany and other countries.

We have reports from England and the United States of lead poisoning due to home fermentation and distillation of wines, beers and ciders in utensils glazed with lead compound. Snuff users have been reported as poisoned; the source being the metallic foil in which the snuff is wrapped. In 1933, 40 cases were reported from Baltimore, with several cases of severe encephalopathy among children; the source here being the use of discarded storage battery cases as fuel. In the same year the same thing happened in Memphis, Tenn., involving five families and 14 children.

Dr. Lockart Gibson of Queensland, Australia, found lead ocular neuritis among a large group of children, due to lead paint on verandas and railings where the children played. Similar cases have been reported in this country.

In 1932 a widespread lead poisoning among mothers and babies was reported in Japan due to lead-containing toilet powder.

In industry the lead hazard is considered greater in the following: lead mining with the exception of galena lead (lead sulphide); lead smelting; lead burning; soldering; tempering; plumbing; buffing and polishing surfaces where lead is an ingredient; manufacturing of lead oxide, lead pigments and or-

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ganic lead compounds; storage batteries; paint; rubber compounding and chemical industry; painting and removing lead paints; enamels and glasses; spray painting; vitreous enameling; pottery dipping; sandpapering, scraping and chipping painted surfaces; flame cutting of painted metal; tree spraying with lead-containing insecticides; type founding; electro-typing and sterotyping.

With lead poisoning so common in industry and the principal mode of entry being inhalation, Dr. Lawrence T. Fairhall, principal industrial toxicologist of the United States Public Health Bureau, has set the permissible content of lead in air breathed by workmen at 1½ milligrams per 10 cubic meters of air. But he is carrying on experiments to determine the permissible content of dusts and fumes of the different forms of lead and will have something definite to report at a later date.

WHAT HAPPENS TO LEAD WHEN IT IS ABSORBED INTO THE SYSTEM

Up until some 15 years ago the prevailing opinion was that lead was transported in the blood stream as an albuminate. Experiments carried out by Aub, Fairhall, Minot and Reznikoff convinced them that lead was carried in the blood plasms as a fairly soluble di-lead phosphate and precipitated in the bones as the very insoluble tertiary lead phosphate.

Aub states in his book on lead poisoning that definite investigation of the form in which lead is transported in the blood is practically impossible due to the very minute amount present, even during an attack of lead poisoning. However, that most of the lead is carried in the plasma has been proved by Aub and his associates. They found in a rabbit with lead poisoning the total lead in the blood amounted to .75 mgm. of which .59 mgm. was in the plasma and .16 mgm. was in the red cells. To show the minute quantity of lead actually in the blood stream even during a severe attack of lead poisoning, Aub found only 2.8 mgm. of lead per liter in a fatal case of lead ENCEPHALOPATHY in man.

To show the distribution of lead in the body after the different modes of entry and after varying lengths of exposure, I will enumerate a few of the experiments relative to this subject carried out by Aub and his associates.

A rabbit was given an intravenous injection of lead and killed in 4 hours. The skeleton contained 13%; the liver 49.3%.

A cat was fed 2.58 grams of lead through the gastro-intestinal tract for a period of 18 days, then killed. The skeleton contained 41.4%; the liver 21.5%. The lead not recovered was excreted through the bowel, never having been absorbed.

A cat was killed 19 hours after insufflation of 5 c.c. of lead carbonate suspension. Total lead in body recovered exclusive of lungs 26.62 mgm.; skeleton 93.6%; liver 4.3%; gastro-intestinal tract 2.1%.

A cat was killed 96 hours after insufflation of 4 c.c. lead carbonate. Total lead in body recovered exclusive of lungs, 72.40 mgm; skeleton 98.5%; liver 1.5%.

So we see that after a very short space of time, 96 hours, the lead is practically all found in the bones where it has accumulated from the lead originally in the liver and other tissues.

Dr. Aub, in his book on lead poisoning, reports 7 human autopsies in cases representing different types and stages of plumbiam. I will mention two of these cases in order to show the similarity of these cases to the animal experiments.

Case 1—A mentally defective child with a perverted appetite gnawed the paint from her bed for several weeks. She developed all the signs and symptoms of lead poisoning. She finally developed convulsions the day before death. Autopsy showed the liver contained 22.99 mgm; skeleton 195.8 mgm.

Case 2—A man, aged 42, had been exposed to lead since childhood when he watched his father spray trees with arsenate of lead. For 27 years he worked in the forestry department himself, using lead arsenate as a spray. During each spraying season he had severe lead colic. Double wrist drop had persisted for 12 years, in spite of treatment. During the last three years there was difficulty in walking. After a month's illness death was caused by pneumonia in mid-winter several months after the last exposure to lead. Autopsy showed lead content in liver .23 mgm.; skeleton 390 mgm.

LEAD ABSORPTION

In industry lead enters the system through the respiratory tract; this means anywhere from the nose down through the lungs, because heavy metals are absorbed just as well through the nose and nasal pharynx as through the lungs, through the gastro-intestinal tract, and very slightly, if any, through the skin.

It has been proved that lead absorbed through the respiratory tract is ten times more toxic than when absorbed through the gastro-intestinal tract. The reason is that a large portion of the lead taken by mouth is not absorbed at all but passes through the bowel with the feces. The portion absorbed from the gastro-intestinal tract is first carried in the portal blood to the liver, which is the most important organ in the elimination of heavy metals.

A large part of the lead carried to the liver is excreted into the bowel with the bile and eventually excreted with the feces. Only a small portion of the lead that reaches the liver is absorbed into the circulation. This explains the late appearance of toxic symptoms following gastro-intestinal absorption.

On the other hand, when lead is absorbed through the lungs, eventually all of it goes directly into the circulation, scattering all over the body to the various parts that are injured by it, including the central nervous system and the kidneys.

A. S. Minot carried out the following experiments on this angle. Cats were prepared by aseptically ligating the esophagus to render swallowing impossible. Suspensions of finely divided lead carbonate, in sterile physiological salt solution were then introduced into the trachea as near the bifurcation as possible. After a few hours the cats were killed

and the amount of lead present in all the tissues, except the respiratory tract, was determined. 20 to 50 mgm. were found. While in another experiment a cat was given daily for 9 days 2000 mgm. of lead acetate solution by mouth. At the end of that time it was killed and only 5.8 mgm. were recovered from the body exclusive of the gastro-intestinal contents.

Absorption of lead through the skin is negligible. However, there are numerous records of cases of plumbism resulting from the application of lead preparations to ulcers, burned areas, skin eruptions, as vaginal douches and eye washes. The fact that lead is not absorbed through the intact skin should not justify any relaxation in the regulations for personal sanitation among lead workers. The hand may still be the means of carrying lead compounds to the mouth or nose where ready entrance to the organism is offered.

At this point it might be well to mention the possibility of lead absorption from leaded gasoline among gasoline station operators and automobile mechanics.

Robert A. Kehoe, Frederick Thamann and Jacob Cholak carried on some experimental work relative to this question in the Kettering Laboratory of Applied Physiology at the University of Cincinnati, and their conclusions were "There is no reason to believe that the usual type of automobile service and repair work involves danger from lead absorption."

SIMILARITY OF LEAD AND CALCIUM STREAMS

Aub and his associates found after extensive research on animals and humans, that the direction of the lead stream is similar to that of the calcium stream. When calcium is being deposited in the bones, so is circulating lead deposited in the bones. When calcium is being pulled from the bones, lead comes with it into circulation and is excreted or re-deposited as the case may be.

In their experiments, the patients were all hospitalized and in special wards in charge of a special metabolism nurse. The feces and urine were collected in 3-day specimens and analyzed for lead by the Fairhall method. With one or two exceptions the lead excretion increased along with the calcium excretion. The amount of lead in each specimen was, of course, small in comparison to the calcium because the relative amount of lead in the human body, even when dying of lead poisoning, is small. Concerning this, Dr. Aub said, "An adult dying of lead poisoning probably has not more than 1 gram of lead stored in the entire body and nearly all this is in the skeleton."

In every case of lead poisoning, the excretion of lead ceased when storage of the calcium in the bones was brought about. This has all been confirmed by research in this country and Europe.

To illustrate this point I will mention two cases reported by Aub in an article in the *American Medical Journal*, Jan. 12, 1935.

Case 1—A man with lead poisoning was excreting nearly 4 mgm. of lead in the feces and less than 1 mgm. in the urine. He was given a high calcium diet plus calcium. His lead excretion ceased. In a few days he was started on gradually increased amounts of ammonium chloride. When he reached his largest dose of ammonium chloride he was excreting 2 mgm. lead in the feces and less than 1 mgm. in the urine and 2 grams of calcium.

Case 2—A young man removed from all lead exposure for 18 months. He had a toxic amblyopia but had received no previous treatment for lead poisoning. Before treatment he was not excreting any lead. He was placed on a low calcium diet for 3 days. At the end of that time he was excreting 1 mgm. of lead in the feces and .5 gram of calcium. He was given ammonium chloride for 3 days. At the end of that time he was excreting 3 mgm. of lead and 1 gram. of calcium. Then he was given parathyroid extract for 3 days. At the end of that time he was excreting 2 mgm. of lead in the feces and 1.5 grams of calcium with a very small amount of lead in the urine.

BONE METABOLISM

Aub and his associates published their studies of calcium and phosphorus metabolism; and of the bone trabeculae as a readily available reserve supply of calcium, in the *Journal of Experimental Medicine*, January, 1929.

They divide the bones into two main parts; first, the hard cortical bone which acts as the body's support with a relatively small blood supply, probably metabolizes at a slow constant rate; second, the trabeculae whch are scattered through the marrow and more numerous at the epiphysis. This relatively small lace-like trabecular structure has a large blood supply; it is here that the available calcium is stored. It is from this storehouse that the body gets its supply of calcium in time of need, is refilled when calcium is stored.

They showed by analysis that lead was stored in high concentration in the trabeculae. Behrens and Bauman showed the same with their X-ray pictures of radio active lead deposits. Vogt, in the *American Journal of Roentgenology* in 1930, also showed X-ray pictures of the deposit of lead in the epiphyseal line of children's bones. In these pictures he also showed where the lead was mobilized and re-deposited.

LEAD EXCRETION

According to Aub, 10% of the excreted lead is excreted by the kidneys, the remainder by way of the bowels. A great deal of research has been done and is still being done in an effort to determine the amount of lead that is excreted by the average individual under normal living conditions and without any exposure to the lead hazard.

Dr. Fairhall told us last spring at the lead conference in Chicago that he thought that .02 of a milligram per liter of urine was about the normal lead excretion for an individual not exposed to lead and living under normal condtions, while a person having lead poisoning may have as much as a half or a whole milligram per liter of urine. At that time he had several experiments under way for the United States Public Health Service to determine not only the normal excretion of lead, but to determine the excretion of lead in lead poisoning caused by the different forms of lead. I expect we shall soon have a report on these experiments and we will know what the official limits are, as far as the

United States Public Health Bureau is concerned.

Kehoe, Thamann and Cholack published a series of articles relative to their extensive research work on the normal absorption and excretion of lead and the absorption and excretion of lead in industry.

In their search for material for determining the normal absorption and excretion of lead, they had in mind comparing a group of individuals living under conditions similar to that of primitive man with a like group living under normal civilized conditions.

For their primitive man group they went to Mexico and located in two little country towns some 50 kilometers northwest from Toluca. They studied 53 subjects in one town and 42 in the other. They analyzed all water and food ingested and all excreta for lead.

Another group was studied in this country, selected at random from individuals never exposed to any lead hazard.

In both instances it was found the amount of lead excreted compared to the amount ingested. However, the amount excreted by the American group was greater than that of the primitive man group because of the higher lead content of the American food.

They estimatetd that the Mexicans ingested .1 of a milligram daily and the Americans ⅕ to ⅓ mgm. They estimated that the normal adult American excretes from .02 mgm.to .08 mgm. per liter urine daily with .03 mgm. to .1 mgm. per gram of ash in the feces.

They estimated a lead exposure as safe if it does not give a lead excretion of more than .6 mgm. per day in the feces and .15 mgm. per liter of urine. If an individual excretes daily lead in the feces at the rate of 1.1 mgm. and .21 mgm. per liter in the urine, lead poisoning may be expected.

Mills Bldg.

Remember . . .

the time is drawing near

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D. J. vs. M. D.

So much has been said about the recent action of the Department of Justice in dragging the medical profession of America into a criminal prosecution, that most of the angles of that peculiar bit of business have been explored. But there must occur to many men certain new thoughts.

There seems to be a sly campaign afoot to discredit anything and everything heretofore held decent in the eyes of the American public in spite of hell and high water. Not too long ago a vast upheaval took place in Washington and when all was waxing merry there appeared on the ash heap of the Administration's disapproval the battered bodies of American business men. Some time later the bar and the press came in for a lot of shoving around and pushing in the face on the part of the Washington Messiahs. Gentlemen in those vocations are still trying to scrape the mud off their togas. Senator Minton, of Indiana, is presently trying to get set in his aim at the seat of the tattered pants of the press gentry of the land. And even yet the lawyers are held up for egg-spattering at various intervals. The American clergy were queried, you remember, on their reactions to the New Deal. But the ministers of the republic said many things that burned Washington faces and they were dropped forthwith from the list of those to be saved by the new millennium. So it wasn't too much of a leap from the hounding of these branches of human endeavor to the straddling of the sore back of the American physician.

Who would ever have thought that old Doc Jones, the family physician, was such a sly devil under his rather kindly exterior? Just imagine the black thoughts in his mind when he wrapped little Johnnie's cut finger. Can't you just see the criminal intent in his eyes when he delivered your own baby? What dastardly thoughts were fermenting in his mind when he gave that antitoxin that saved the neighbor's kid from death by choking that winter diphtheria swept the village? What clever scheme was he pondering that night he got your appendix? What horrid plans did he have when, in trying to wipe out small pox, he fought for compulsory vaccination in his neighborhood?

It must be a great surprise to the American public to now learn that the doctor whom they have trusted thru a life time is a blackened criminal, scheming and conniving against their welfare. Indeed Jehovah, Himself might be surprised in this case.

Well, of all the — —!!

YOUR ELEPHANT RIDE

When poorly-veiled slurs are cast on the American Medical Associaiton by irresponsible scribblers of the nation's press, just who are they slurring? When quacks and cults assail the American Medical Association, which they bitterly hate and fear, just who are they assailing? Are these battles being pitched in the camps of one or two men attached to the Chicago headquarters of the A.M. A.? When some professional brother says that the A. M. A. is not doing right by him, just who is he talking about? He may think he is annoyed with one or two men in the Chicago headquarters, but is he really?

It is pertinent to restate that the American Medical Association is you and me, that our Chicago officials are only in office at our pleasure, to carry out mandates and orders given them by you and me. When the A. M. A. is maligned by any critic he is simply talking about you and me. It would seem to be the democratic thing and the intelligent thing to understand this. There is no mysterious dictatorship or bureaucracy in Dearborn Street. The policies enunciated by the national officials of the American Medical Association are but reflections of what you and I will and decide. Dr. Nathan B. Van Etter, speaker of the House of Delegates of the American Medical Association, had this to say at the San Francisco session this summer:

"The American Medical Association is the largest medical organization in the world. Its technical setup is democratic by any test. Any member, anywhere, at any time may express himself on any subject with the greatest freedom before his county medical society and then to this House of Delegates for decisive action.

Representing, as you do, all of our states and territories, the changes are not likely to be revolutionary responses to the excitement of occasional individuals among the more than 109,000 members, or servile responses to inspired propaganda. When county societies become interested in new medical service plans and transmit their ideas to state societies, which in turn send them to your sessions, you will discuss them as you have always done, and when a majority of you approve them they will be adopted and carried out by your executives.

You elect the officers and trustees and they re-

port to you. They do not originate policy. You make the policies, and the officers and trustees are obligated to execute them. If your policies are not carried out to your satisfaction, you may replace your officers. On your shoulders rests the fate of American medicine. Every practitioner of medicine looks to you, and back of these practitioners are millions of people who are daily looking for the best in medical care, not merely adequate care or just any medical care that may be promised by paternalistic bureaus, but a constantly improving medical care that a constantly improving medical profession may offer them.

The House is composed of 175 delegates. There is no dictator among your officers. There is no dictator among the trustees to whom you delegate the care of your money. There is no policy which you may not reverse. You have the power and you have the responsibility. As delegates from constituent bodies it is your duty to report the action of this House to your membership. Judging from the lack of knowledge of the association among physicians, and judging from the amount of misrepresentation about the American Medical Association which constantly appears in public print, it seems evident that you do not sufficiently exercise intelligent interpretation of the association to your members at home.

If this great association is a blundering elephant, remember that you are riding him.

If this great association is unresponsive to the will of its membership, remember that you may have failed to bring the thought of your constituents to this forum."

BLOOD TRANSFUSION

The possibility of transferring blood from the body of one animal into that of another began to intrigue medical men shortly after the discovery of the circulation of the blood by Harvey in 1616. In 1667, Denys, physician to Louis XIV, recorded the transfusion of the blood of a lamb into a small boy. Blood transfusion fell into some disrepute rather soon thereafter, because types and species incompatibilities were not understood. Therefore, the physician never knew when he was apt to have violent reactions and even death follow attempts at transfusion. Following the work of Jansky and Moss in the early part of this century on the agglutinins of blood, a wide field of therapeutics unfolded.

Not so long ago blood transfusion was usually an emergency measure. It was a weapon at the command of large hospitals and research centers. The practitioner out in the field rarely had recourse to this potent aid; but today the procedure is used from the cross roads to the metropolis.

Until the work of Yudin[1], of Moscow, was published there were two principal methods of transfusion. They were the direct uncitrated and the indirect citrated procedures. Yudin, however, began to work with the transfusion of cadaver blood which had been stored under refrigeration over a period of several days to weeks. In this method blood was taken from corpses shortly after death, was typed and tested for syphilis, then stored for future use. Remarkably good results were obtained following transfusion of this blood. So that now it is an accepted method in several of the larger clinical centers in America.

Blood transfusion is indicated in:

(1) *Hemorrhage;* where it may be a life saving measure following loss of blood from such emergencies as ruptured ectopic pregnancy, gastric ulcer, postpartum hemorrhage, severe traumatic injuries, the hemorrhogic diatheses as purpura hemorrhagica and hemophilia.

(2) *Septicemia;* where fresh antibodies are of value in both acute and chronic types of sepsis.

(3) *Shock;* transfusions are of particular value where there has been a sudden loss of blood.

(4) *Immunotransfusion;* for such diseases as scarlet fever, typhoid fever, brucellosis and postvaccinal encephalitis.

(5) *Malnutrition;* such as in infants with digestive disturbances and diarrhea.

(6) *Blood dyscrasias,* such as leukemias and various anemias.

(7) *Carbon monoxide poisoning;* where fresh blood is needed to carry on oxygenation.

Despite the wide-spread use of blood transfusion and its ease of performance, there are certain warnings as to possible complications or difficulties:

(1) If the continued drip method such as employed in the Bradford Memorial Hospital in Dallas in 1933 is used, care must be taken that tubes and cannulae do not become clogged from sedimentation of the corpuscles.

(2) The transfusion should not be given too rapidly.

(3) Donor and recipient should be compatible, being cross-matched for sub groups.

(4) Equipment should be scrupulously clean, chemically neutral and sterile.

(5) The absence of such blood-borne diseases as syphilis, malaria and bracellosis should be established in the donor's blood.

(6) The need of transfusion should be anticipated long before an emergency arises.

(1) Yudin, S. S.: Transfusion of Cadaver Blood (1000 Cases): S. A. M. A., 106:997, March. 21, 1936.

A. M. A. SURVEY

For a long time too many individual physicians have not taken active interest in the affairs of their medical societies. It would seem that this lack of participation in the economic affairs of the medical societies should be ended forthwith. The presence and the strength of all members of the American Medical Association is needed now. Some time ago the Association inaugurated a nation-wide survey of the medical needs of the American public. This survey took the form of individual questionnaires. For the success of the survey to be established it is necessary that a complete response be obtained from the entire membership of organized medicine. It may be true that the reports and figures of one physician are but a drop in the bucket, but the bucket must be filled. So that officials whose task it is to correlate the results of the survey are requesting early completion of the blanks which were given to physicians several months ago. Conclusions to be drawn from this nation-wide study will probably not be available for several months. Here is a chance for each individual physician in America to contribute his bit in a vital group undertaking. It is urged that every physician of the Southwest send his completed survey blank to his headquarters immediately.

Special Section

Arizona State Medical Association

J. D. HAMER, M. D.
Associate Editor

MEDICAL ASSOCIATION ACTIVITIES

Attention of the membership is called to the fact that there is to be a Special Session of the House of Delegates of the American Medical Association for September 16 at Chicago, Illinois, the session to continue until its business is concluded. The Official Call for the Special Session stated that—Quote—"The business to be transacted at this special session shall be limited to the consideration of the National Health Conference recently held in Washington and such other matters as may be submitted to the House of Delegates by the Board of Trustees".—Unquote.

The national health program referred to embraces the following:

1. Compulsory insurance with federal subsidy.
2. Expansion of general public health service, requiring additional annual expenditure of $200,000,000.
3. Expansion of maternal and child health services requiring additional annual expenditure of $165,000,000.
4. A hospital construction and partial maintenance program to construct 360,000 beds and 500 health and diagnosis centers, requiring annual appropriation of $146,050,000.
5. Expansion of facilities for medical care of the medically needy to reach an annual appropriation of $400,000,000.

The program as set forth would increase in a ten year period, at which time estimates would reach a maximum annual cost to the federal and state governments of $850,000,000. This does not cover the compulsory insurance item. The authorities suggest a minimum of $17.50 per person a year as the cost of furnishing adequate care, exclusive of dentistry.

It is with this program that the special session of the House of Delegates will concern itself. Attending from Arizona will be Dr. J. D. Hamer, Phoenix, official delegate of the Arizona State Medical Association, and Dr. D. F. Harbridge, Secretary of the Arizona State Medical Association, who has also been summoned for the purposes of the session.

Obituary

DR. GEORGE E. GOODRICH

On Saturday, September 3, 1938, at Los Angeles, California, where he had gone to spend the summer months, Dr. George E. Goodrich, of Phoenix, passed away of a heart ailment. For 31 years Dr. Goodrich had been prominent in the practice of medicine in Arizona. For many years he was chief surgeon for the Arizona Copper Company at Morenci, and participated actively in the business and civic life of that community. Dr. Goodrich relinquished his post at Morenci to assume a similar one with a coal mining company in Illinois, which was his native state. Arizona had cast its spell over him, however, and he returned to enter private practice in Phoenix, where he became prominently identified with its civic and business activities. He served the Phoenix Chamber of Commerce in an active capacity for many years and was a member of the Phoenix Country Club and the Arizona Club.

The medical activities of Dr. Goodrich included the following: Superintendent of Public Health under the administration of Governor Thomas Campbell; member of the Medical Advisory Board for the Selective Service System during the World War for which he received citation from President Wilson; Chief Surgeon of the Arizona Copper Company at Morenci, and member of the Arizona State Medical Association. In these various capacities he rendered outstanding service.

Dr. Goodrich's associates extoll him for his professional principles and for his kindliness of character as well as for his professional skill. The Arizona State Medical Association shares with his immediate associates and his family a keen sense of loss in his passing. Interment was at DeKalb, Illinois, his birth place, on Wednesday, September 7. Mrs. Goodrich survives him.

LYMPHOSARCOMA

Presentation of a Case

by
M. W. MERRILL, M. D.
Phoenix

In the presentation of any case, one should be sure that such will add in some small measure to the knowledge of a condition. I am presenting a case of lymphosarcoma which is interesting from several standpoints. First, this case has been more or less under our observation since the first definite symptoms appeared. It was diagnosed by biopsy, received the accepted treatment, and after expiration, an autopsy was performed. It is interesting also because this case was considered one of tuberculosis for a period of about three months. Then again, although typically a lymphosarcoma, the histologic picture is not clear-cut in this regard. The true diagnosis, when it was finally made, was so late that the maximum benefits from treatment could not be obtained.

Some think it is perhaps futile to spend much time studying those cases for which we can do no more good than we can for lymphosarcoma and related conditions. However, it is well to bear in mind that we must constantly study these cases if we are ever to be more successful in diagnosing and treating the disease.

The essential etiology of lymphosarcoma remains unknown. Dr. A. U. Desjardin of Rochester, Min-

nesota, considers chronic inflammation as the most important single factor in the production of malignant growths of lymphoid tissue. Familial tendency plays perhaps a small part. Tuberculosis as a factor has been debated pro and con, but a definite relationship has not been established. The general symptoms of these conditions vary greatly with the site of the lesion, the rapidity of its progression, the extent of development and rapidity of ulceration and necresis, and the relationship to important and vital structures.

Dr. Max D. Cutler in a comprehensive review of 30 cases treated in the Michael Reese Hospital in Chicago, studied over a period of two years, found the average duration of the disease to be 16 months. The minimum duration, two months, and the maximum duration four years and six months. At the time of his write-up, 57% of the 30 cases had succumbed to the disease. He found lymphosarcoma more common during the fourth and fifth decades of life, although it may occur in older or younger individuals. It is more common in males than in females. Cutler differentiates two forms of the disease; a generalized condition which is most common, and a localized form. Prognosis is better in the localized form when it can be diagnosed early and given adequate X-ray treatment.

The differentiation between Hodgkin's disease and lymphosarcoma is of academic, if of no other interest. Dr. Solomon Ginsburg of New York, who has been studying lymphatic growths for a number of years, is convinced that, biologically, the two diseases are the same. Ginsburg believes that, taking into consideration the variations due to different environmental and constitutional conditions, the diseases can be classified as one and the same. However, leading pathologists differentiate the two conditions histologically in most cases, and the generally accepted view is that lymphosarcoma runs a more acute course, progresses more rapidly to a fatal termination, more readily necroses and infilterates surrounding tissue, and in general presents more of those characteristics of a malignancy. Lymphatic leucemia must also be ruled out, as it may similate these conditions.

Aside from differentiating between Hodgkin's disease and lymphosarcoma, it is well to bear in mind, as Ginsburg points out, that both of these conditions, instead of being localized affairs, are protein in character. Lymphosarcoma or Hodgkin's disease may metastisize to practically any other tissue in the body.

Treatment of lymphosarcoma can be summed up as follows: Early diagnosis, adequate X-ray therapy, and good general clinical management of the patient. Prognosis is of course very bad. Dr. Lloyd F. Craver, in a recent study of 96 cases, found only seven five-year survivals, and out of these seven only two were without clinical evidences of the disease. These cases were studied at the Memorial Hospital in New York.

The case in question first came under our observation early in December, 1937. At that time he suffered a pulmonary hemorrhage, the origin of which was ascribed to tuberculosis. Hemoptisis continued for approximately 10 days. The patient was then sent to the pathological laboratory, where an X-ray of his chest was made. The report at that time showed a "defnite increase in the size of the ascending aorta and mediastinum. The heart was not increased in size in its transverse diameter. There is abnormal shadow around the hilum of each side and definite density extending out into the lung fields of the upper right lobe well out toward the periphery. These shadows probably are due to tuberculous infection at the hilum spreading into the lung tissue. There is some increase in density around the left hilum but not so marked a degree as on the right." Following this report, the patient was sent to a County Tubercular Rest Home where he remained for a few days. Shortly after this, he was moved out onto the desert northeast of Mesa, where he remained in bed until March, 1938.

At that time he began to complain of some swelling of his ankles and legs and of increasing pain in his chest and right shoulder region. He was brought to the County Clinic, and from there, sent into the State Welfare Sanitorium at Tempe. His chief complaints at that time were loss of weight, gas on stomach, some nausea, and pain in the right shoulder and chest. Laboratory findings, including urinalysis and complete blood work-up, were essentially negative. An X-ray taken at this time showed some peculiar densities in the right mediestinal area and prominence of the aortic arch on the left. After a physical examination by Dr. Lloyd Swasey and a consultation with Dr. Hilton J. McKeown, the case was diagnosed as either a lymphosarcoma or a Hodgkin's disease, and was referred back to the county.

The patient was then admitted to the Good Samaritan Hospital, Phoenix, on March 21, 1938. His complaints were the same as before noted. He was having considerable pain, and morphine was necessary to give him relief. The past history up until two years before the onset of his pulmonary hemorrhage contained nothing of importance. His family history was entirely negative, and no history of any cancer of any kind is known. In the two years preceeding his hemorrhage, the patient lost about 20 pounds in weight, had been bothered by considerable bloating and gastric disorders, and had complained occasionally of a heavy feeling in the right upper side of his abdomen.

Physical examination on admission revealed a white male of about 50 years of age, obviously quite ill. Positive findings were as follows: Edema of the face and neck, palpable masses in the left axilla and supraclavicular region, dilitation of the superficial veins of the right side of the chest, and some distention of the abdomen. The abdomen was tender to palpitation. Lung findings were essentially negative, except for a few rales in the right upper chest. The left chest was negative. Heart examination revealed nothing of note.

Blood pressure was 126/86. Urinalysis was negative: Red blood count 4,280,000; leucocytes 7,600; hemoglobin 77; polynucleus were 84, lymphocytes 10, and mononuclears 6.

Impression was either Hodgkin's disease or lymphosarcoma. On March 28, 1938, under local anesthetic, I removed one of the tumor masses from the left axilla for biopsy.

The microscopic diagnosis of the tumor mass was sarcoma, probably neurogenic in type. Following this report, the patient was again X-rayed with the following findings: "We have made examination of this patient's chest and compared the findings with those shown by film taken December 13, 1937. There has been a marked change in the character and amount of shadow. On the right side, there is an increase in the shadow which shows slightly irregular enlargement well out into the lung field. There is density in both upper and lower portions of the right side. On the left side, there is shown the enlargement of the aorta, both at the arch and in the descending portion. These changes would seem to indicate that this is either a lymphosarcoma arising from the mediastinum, or possibly Hodgkin's disease. The preferable interpretation would be lymphosarcoma, with increase in density throughout the right side, probably due to interference with circulation."

After this X-ray, the patient was started on a series of radiation treatments by X-ray. He re-

ceived a total of eight over the mediastinum and right axillary region. Treatments were completed on April 18, 1938, and the patient was discharged from the hospital considerably improved on April 19, 1938. During his treatments, he occasionally ran a low-grade fever, the first since his admission to the hospital, and complained considerably of nausea. Before the treatments were completed, it was possible to stop morphine and substitute codeine for pain.

On April 19, the patient was removed to the Camelback Rest Home, with instructions to report in one month for further check-up. For a week he did very well. Then his pain recurred, considerable nausea and bloating developed, and during the the next two weeks, his down-hill course was rapid. He became deeply jaundiced, very badly distended, and suffered a great deal, a half grain of morphine being necessary every three hours. He grew steadily worse and expired on May 9, 1938. Permission was granted for the body to be moved to Mesa, where Dr. A. H. Lepon and I performed an autopsy.

On autopsy, no evidence of the axilliary or supra-clavicular mass was noted. The skull was not examined. The right lung presented a marked degree of atelectasis and on section, neoplastic material was found obliterating the bronchi and other air passages. Grossly, the mediastinum presented a growth the size of a grapefruit, which was intimately connected with and compressed the hilum of the right lung. The heart showed no gross pathology. Some arteriosclerotic changes were present in the aorta.

The liver showed congestion and the gall bladder was enormously distended with bile. A large retroperitoneal mass was found which resembled that taken from mediastinum. This mass was located in the retroperitoneal space and its growth involved the bile passage, completely blocking the common duct. It also involved the pancreas and the second and third portions of the duodenum. It extended downward almost to the bifurcation of the aorta. Tumor masses were also found surrounding the left kidney. On section, the specimen showed: "Large masses of atypical round and spindle cells with very numerous mitotic figures. In still other portions there is slight alveolar arrangement. The cells are arranged along the vascular spaces in a palisage-like arrangement of cells somewhat resembling endothelioma. However, the entire picture is more that of a sarcoma, neurogenic in origin."

Diagnosis: Neoplasm of mediastinal glands and retroperitoneal space, obstructing the bronchi of of the right lung, and the common duct.

Summary: In looking backward, it is quite evident that the original hemorrhage came from an ulceration into the bronchi by the tumor. The slight lung findings suggestive of tuberculosis, can be attributed to results of the growth. This case illustrates the value of a careful and complete work-up, and although it was probably impossible at first to make a definite diagnosis, had he been kept under closer observation, it is probable that an earlier diagnosis could have been made and more benefits secured from X-ray. The case was undoubtedly lymphosarcoma, with a histological variation resembling a tumor neurogenic in origin.

15 E. Monroe.

Read before Staff Meeting, Good Samaritan Hospital, March, 1938.

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Special Section

New Mexico Medical Society

L. B. COHENOUR, M.D., Albuquerque
Associate Editor

RULES GOVERNING APPLICATION OF THE FEE SCHEDULE*

1. Reports relative to the condition of the injured employee under treatment shall be made by the attending physician without extra charge.

2. Reports must show in detail the nature and extent of the injury and a full description of the treatment.

3. Bills must be itemized showing the date of each visit, dressing or operation and charge for each service.

4. In cases where flat fees are allowed, it is not permissible to charge additional fees for visits, dressings, treatments, etc.

5. Fees for daily dressing shall not be paid unless the nature and extent of the injury as shown by the proof on file clearly indicates the necessity for same.

6. The fee schedule includes all dressing unless the case shows that an excessive amount of material had to be used, in which case it will be charged at cost.

*Adopted between U. S. Fidelity and Guarantee Co. and the New Mexico Medical Society.

SCHEDULE

First visit to place of injury, including dressing not otherwise specified	$4.00
Night, 10 p.m. to 7 a.m.	5.00
First office visit, including dressing	2.50
Subsequent visit at office or hospital	2.00
Country mileage—one way, beyond city limits of incorporated town, including treatment, dressing	.75
Subsequent home visits, etc.	3.00
M.D. assistant to surgeon at major operation	25.00
Anaesthetics for major operation when administered by qualified anaesthesist:	
Ether	10.00
Gas	15.00
Spinal and Sacral	15.00
These fees do not apply to anaesthetics administered by the operating surgeon.	
Local anaesthetic	2.00

FRACTURES

Flat Fees—Including application of Casts, Splints, Bandages, etc., whenever a fracture occurs at or near a joint and there is also a dislocation of the

joint, the flat fracture fee also covers the reduction of such dislocation. No additional fee shall be allowed for the treatment of cuts, contusions, abrasions, etc., adjacent to the point of fracture.

In case of more than one fracture the fee shall be the major one plus 50 per cent of the fee prescribed for each of the others as herein classified.

Femur, Simple	$100.00
Patella	50.00
Clavicle	35.00
Scapula	35.00
Radius or Ulna	35.00
Radius and Ulna	50.00
Humerus, Simple	75.00
One finger	15.00
Each additional finger	5.00
One toe	15.00
Each additional toe	5.00
Metacarpals—one or more	25.00
Tibia	50.00
Fibula	25.00
Sternum	15.00
Skull fracture of vault, with or without debris	50.00
Basal fracture with dressings	50.00
Tibia and Fibula—or Potts' Fractures	75.00
Ribs—one simple	10.00
Each additional rib	7.50
Metatarsals—one or more	30.00
Pelvis	75.00
Spine—transverse process—one or more	50.00
Spine—body of vertebrae—one or more	150.00
Os Calcis	50.00
Astragalus	30.00
Lower Jaw—not including dental	40.00
Tarsal—other than astralgalus or os calcis	15.00
Fracture of vault or base requiring decompression, trephining or otherwise	100.00

An additional charge of 50 per cent may be added in cases which require open operation for reduction of fractures. These flat fees include preliminary treatment, reduction and subsequent treatment.

AMPUTATIONS

Flat Fee—Including amputation, preliminary and subsequent treatment. No additional fee shall be allowed for the treatment of cuts, contusions, abrasions, etc., adjacent to the point of amputation. In case of more than one amputation the fee shall be the major one plus 50 per cent of the fee prescribed for each of the others as herein classified.

Thigh, leg, ankle, or foot	$ 75.00
Arm, forearm, or hand	50.00
Finger or toe	20.00
Fingers or toes (two or more)	25.00
Arm disarticulation at shoulder	100.00
Hip disarticulation	125.00

DISLOCATIONS

Flat Fee—Including preliminary treatment, reduction and subsequent treatment.

Hip dislocation of head of femur from hip socket	$50.00
Wrist	15.00
Finger or toe	5.00
Shoulder—subsequent dislocation	15.00
Shoulder—primary	25.00
Elbow or ankle	25.00
Knee	35.00
Jaw	10.00
Clavicle	20.00
Clavicle—open fixation—either end	50.00

EYE-SPECIALISTS

Removal foreign body from cornea or conjunctiva	$ 4.00
Removal foreign body from anterior chamber of eye	50.00
Removal foreign body from vitraous chamber of eye	75.00
Refraction	5.00
Removal traumatic cataract	50.00
Cauterization of corneal ulcer—chemical	5.00
Cauterization of corneal ulcer—thermal	10.00
Enucleation without implantation	50.00
Enucleation with implantation	75.00
Iridectomy, including subsequent care	50.00

X-RAY

The following fees are for ordinary examinations and are for a sufficient number of plates to determine the nature and extent of the injury. These fees include interpretation and written opinion, when required. If hospital does the work, arrangements are made with the hospital.

Ankle, arm, elbow, femur, foot, forearm, hand, jaw, knee, leg, and wrist	$ 6.00
Stereos—additional when authorized	2.50
Hip	7.50
Shoulder	7.50
Head, vertebrae, pelvis	10.00
Chest	15.00
A. P. and lateral spine	15.00
Localization foreign body	15.00
Intestinal and stomach studies including screen examination and plates	25.00

Examinations made outside of office, and use of portable X-ray equipment, additional fee of 50 per cent.

Bill for X-ray examinations should always be itemized and should show the various dates upon which pictures were made.

MISCELLANEOUS OPERATIONS

Hernia, radical operation, preliminary and subsequent care	$100.00
Laparotomy, radical operation, preliminary and subsequent care	125.00
Autopsy—when authorized—complete with written report	35.00
Autopsy—attending but not performing—when authorized	10.00
Physical examination by other than attending physician: Amount to be agreed to between Insurance Company and the Physician when examination is ordered.	

L. B. COHENOUR, M. D.,
Sec'y. N. M. Medical Society.

MISCELLANY

OPTOMETRY ON TRIAL

A recent number of the Readers' Digest contains an article entitled "Optometry on Trial." The author, Roger William Riis, in masterly style describes how patients with normal eyes were fitted with glasses by a number of different optometrists with prescriptions that varied widely, while those with defective vision who had previously been examined by thoroughly competent oculists, received considerably worse treatment from optometrists than did normal patients, not in one section of the country but in widely different regions. He went on to describe how the eye signs of a constitutional disease, which any competent oculist would recognize, were completely missed. This article has received wide discussion not only among the medical profession but among the laity. It will accomplish far more toward seeing that this bespectacled nation receives proper eye examination than any amount of shouting from the housetops indulged in by the oculists themselves or the medical profession.

We cannot thank this author and the Readers' Digest enough for the wholesome publicity which they have given to this subject. But, if we wish to take advantage of this unasked for, unsolicited, and unpaid for help, we must first put our own house in order.

Patients have been referred by licensed physicians to optometrists for eye examinations, to commercial laboratories for x-ray examinations, to so-called pathological laboratories run by technicians, and to non-medical men for physiotherapy and manipulation. There is no royal road to health, and the fare is not cheap. First-class, adequate medical attention by the general practitioner or in the specialties, is costly. Somebody must bear that cost. In many cases the physician partly bears it by reducing his fees. In other cases the community contributes; but no matter who pays, it is still costly goods. So-called cheap medical

care is often most expensive in the time that may be lost and the damage that may be done, or in the failure to recognize the true road to renewed health and strength. No one should recognize this better than medical men. There should be no false economy on their part to try and save their patients a few cents or dollars by employing cheap assistants or by referring their patients to those poorly qualified to give auxiliary services. In some few cases, no doubt, this reference has been deliberate with the idea that, if the service costs less, the patient would have more money left to pay the referring physician. We might as well admit that this has sometimes been the motive.

For the benefit of both the patient and the ultimate good of the medical profession, medical services of all kinds should be in the hands of medical men themselves. Then will we really be entitled to such help from the laity as we have received in the above-mentioned article.

—*J. of Med. Soc. of N. J.*

GAME OF "DIVIDE, AND RULE"

The first of the Caesars did very well for himself and his party by putting action to the maxim: "Divide, and Rule." With varying degrees of success, the rule has been followed in every age and in every land since the Roman conquests. It is still being employed, in America and elsewhere, with just as much efficacy as in the year one, A.D.

The field of American medicine is not immune from attempts by some to utilize this very efficient proverb of the centuries. It seems apparent that a determined program has been developed to create civil war in organized medicine, for the purpose of weakening the unity of an efficient profession, and then to seize the reins and subjugate medical practitioners.

We read of attacks made by leaders of specialty groups; metropolitan papers play up the "Declaration of Independence" of a small minority committee; magazine articles by the score heap undeserved opprobrium on an altruistic profession. All to what purpose?

Is some force inside or outside the ranks of medicine attempting to divide, and rule the profession? Are these vitriolic attacks the result of a carefully conceived campaign to develop a defeatist attitude among practitioners, and then quickly to foist upon the people a system of practice not in accord with American traditions and design of living? Are the medical figureheads in the van of attack being used as dupes by this force?

Doctor, you have made the answer. In fact, you and the many other thousands of members of organized medicine *are* the answer. There will be no repetition of "Divide, and Rule," so far as American medicine is concerned, if you remain constantly aware of current events, if you stand united against attacks within and without by active support of your County Medical Society, and if you not only know the truth but spread your knowledge to the people—day in and day out, every month of the year.

—*J. Mich. St. Med. Soc.*

ANOTHER TRY AT DIAGNOSIS

With its June issue, the *American Magazine* joined a constantly growing list of periodicals devoting editorial space to what they apparently believe is a matter for grave public concern—the practice of medicine in the United States. Its contribution was an article authored by one Beverly Smith, under the title, "Diagnosing the Doctors."

Like most other articles of the kind, it seemed characterized principally by a desire for some kind of change, although without any great certainty as to just what kind of change is needed. It suggested, by citing instances, the desirability of fixed-fee clinics caring for certain groups of people, but as usual, neglected to point very clearly why similar systems might not work well under conditions existing among other groups. It suggested the so-called hazards of specialists fees, without suggesting very many alternates that would prove entirely satisfactory.

It pointed out that the public "is willing to pay more, in total, for medical care than it does now, if the burden is more fairly distributed, and if the doctors will organize themselves to give more efficient service." It would be interesting to know just how the public believes the burden can be more fairly distributed—and what it believes constitutes efficient medical service.

And to emphasize the horrors of being financially unprepared for sickness, the story is told of a farmer who was practically bankrupt by rheumatism, in spite of the fact that his family doctor apparently suggested early in the game that he should go to the city to seek the help of specialists whose ability in such cases the general practitioner recognized as superior to his own. But the farmer "*kept putting it off.* Afraid of the fees. Finally he got so crippled he did go to the city." As in so many other cases of "putting it off," fear of fees may sound logical, but does it tell the whole story?

Perhaps not, but articles like this, appearing with increasing frequency in the lay press, are bound to force a move from the medical profession, and perhaps a good preface to any move would be a broader attempt to tell, where the public will see, read, and hear it, the doctor's side of the story.

—*Victor News.*

PUTTING THE SCREWS POLITELY ON THE DOCTORS

The New Deal's Department of Justice has moved against the American Medical Association as a violator of the anti-trust laws. Its offense, in brief, is that it has expelled one of its members who cooperated with members of the staff of the Group Health Association, an organization of government employees formed for treatment of its subscribers. It is also alleged that Washington hospitals have been shut against the doctors of the group.

A reading of the opinion of the department would be proof that Attorney-General CUMMINGS did not write it. Instead of his trustbusting style it has the soft, even pussyfoot, approach of his assistant, THURMAN ARNOLD. Professor ARNOLD, while under orders to shake a club at the medical association, possibly remembered that some of its members, when they could reach the polls without neglecting the critical case of Mrs. Jones, voted for the New Deal. So he takes pains to explain that the offenses committed are not "crimes which reflect upon the character or high standing of the persons who may be involved." They have no more moral turpitude than a reckless driver who is a person of "distinction and good will in a hurry to meet his legitimate engagements." However, the Professor explains, the law says so and so and the Department of Justice must follow up.

The accused doctors may keep their silk hats on. They may also reflect that labor unions and farmers may combine but physicians must not do that. They may remember, too, that there is a strong current in the New Deal toward socialism, including socialized medicine. Professor ARNOLD's lecture on the high cost of medical treatment should amuse at least one veteran practitioner of this town who had 130 calls in a recent month and collected a total of $3 for his pains.

But the Washington doctors may as well prepare to be indicted. Their lawyers can read to the jury some of ARNOLD's touching assurances of their lack of moral turpitude.

—*New York Sun.*

THE SOFT IMPEACHMENT

The nation is just getting itself adjusted to Mr. Thurman Arnold's novel method of anti-trust prosecution by what might be called the soft impeachment. His recent announcemnt that he was setting out to enjoin the motion-picture companies as violators of the law, but was doing so in only the most constructive and friendliest possible spirit, had much to recommend it; here at last, it seemed, was a way through which the anti-trust laws might be made into really serviceable instruments of rational industrial regulation. But when Mr. Arnold suddenly extends the same polite constructivity into the non-industrial problem of group medicine one is not, in the first moment of astonishment, quite so sure.

The implications of the proceedings are so remarkable as rather to overshadow the specific allegations of sabotage against the district medical society. To begin with, Mr. Arnold can scarcely have much confidence that a criminal prosecution would stick in the courts. There are various decisions to the effect that the prohibited restraint of "trade or commerce" must apply to goods or commodities, while the Clayton act specifically excludes the "labor of a human being" as an article of commerce. Surely, a doctor's laborious services could not easily be brought within the scope of the anti-trust laws. But the Arnold method is not aimed at a court battle; it proposes rather to extort a consent decree without one.

The consent decree has been used before now to enforce, through the mere threat of prosecution, an extent of governmental regulation which the prosecution itself might fail to sustain. That aspect of Mr. Arnold's sweet reasonableness might not matter so much in the proper field of anti-trust law, where there are always plenty of capable opposing counsel; but extended into the problems of medical practice it seems fraught with ominous possibilities.

And there are other peculiar things about this proposal to settle the complex social issues centering around medical care by the kindly intervention of the Department of Justice, with criminal charges and a grand jury investigation. Mr. Arnold himself notes, under the head of "economic results to be expected" that prosecution in the nation's capital will insure "adequate publicity." (It would insure little else; for, since medicine, if "commerce" at all, is certainly not interstate commerce, the Federal law would operate only in the District of Columbia.) At a momentwhen the New Deal seems pretty hard put to it for popular issues next year, and is clearly working up the health insurance question as a possible means of filling the gap, this device for showering "adequate publicity" upon the matter has an unpleasantly political connotation.

The Department of Justice, for all its excellences, is clearly not the appropriate agency for regulating American medicine. And if its new policy under Mr. Arnold is going to be bent to the uses of political stratagem, it will cease to be an appropriate agency for regulating anything else.

N. Y. Herald Tribune.

DOCTORS AND US

The average layman holds himself aloof from the controversy about his welfare now raging between the Federal Government and the American Medical Association.

If he is a middle-class citizen, he considers himself a martyr, and is fond of saying that only the very rich and the very poor receive any consideration from doctors—although I doubt whether he would be willing to exchange places with the poor.

If we are honest, we must admit that our attiutde toward the doctor has always been irritable and without consistency. We're ready to spend money on everything except our health.

Most families will make any sacrifice to buy an automobile on the installment plan, while they feel much aggrieved if they have to invest monthly sums to pay for medical attention which may have kept one of them out of the grave.

Women complain about the awful doctor's bills—and there's no denying that they look enormous when you are meeting them—while in the very next breath they tell you about the lovely little frock they picked up yesterday for only $39.75, or offer you some of the Scotch hubby bought yesterday, or let you in on their plans for vacationing in Honolulu.

No, friends, there's no excusing ourselves. If we had always paid the doctor as willingly and as promptly as we pay the dressmaker, the liquor dealer and the garageman, some of our medical problems wouldn't be haunting us now. Perhaps socialized medicine would not stare us in the face.

A woman I know expressed what I mean when she said:

"I was good and mad when the bill for my operation came—$250. It seemed preposterous. I couldn't put all that down in a lump sum, so I resorted to installments.

"As time passed I felt better and better, more like my old self, and then one day as I was writing the check the thought struck me—no money can ever really pay for what the doctor has done for me. He has created a new well woman out of the old worn-out sick one. He's made me feel a zest for life again. He's separated me from all pain.

"Then I saw how little and mean I was because I felt unwilling to sacrifice luxuries for good health and perhaps many more years of life."

—*MRS. WALTER FERGUSON,*
in Scripps-Howard Newspapers.

RABIES: REPORT OF TWELVE CASES, WITH DISCUSSION OF PROPHYLAXIS

MAURICE L. BLATT, SAMUEL J. HOFFMAN and MAURICE SCHNEIDER, Chicago (*Journal A. M. A.*, Aug. 20, 1938), discuss the twelve cases of rabies admitted to the Cook County Hospital between 1929 and 1937. All proved fatal. The diagnosis in each case was confirmed by necropsy. The incubation period for the patients varied from two weeks to two months. The closer the site of the bite to the central nervous system the shorter was the incubation period. Wounds made by the bites of animals should immediately be cauterized with nitric acid. The Pasteur treatment or one of its modifications should be instituted in accordance with rules outlined and accepted. The twelve persons whose cases are reported died after suffering great agony and might have been saved if adequate prophylactic measures had been instituted immediately. They were admitted to the hospital after having been ill from two to seven days and anywhere from two weeks to two months after they had been bitten by dogs. Stringent enforcement of regulations governing ownership, licensure, muzzling and leashing of dogs would have prevented the bites. The extent of this problem is evidenced by the fact that in the state of Illinois alone 18,466 dog bites were reported to the state department of public health in 1936 and that there were ten deaths from rabies. A knowledge of similar facts would divulge a tremendous loss of time and lives of human beings and animals in the United States from a preventable cause. When such knowledge becomes public, it will be of

inestimable educational value in the eradication of this dreadful malady.

VISUAL STANDARDS FOR OPERATING MOTOR VEHICLES

Recognizing the increasing necessity for greater care in the operation of motor vehicles on the highways, the House of Delegates of the American Medical Associaton at the recent San Francisco meeting adopted the following resolution. The standards set forth were developed by the Section on Ophthalmology, where this program had been under consideration for many years:

Resolved, That the following be accepted as the approved American Medical Association standards:

A. For an Unlimited License:
   1 Visual acuity with or without glasses of 20/40 Sn. in one eye and 20/100 Sn. in the other.
   2 A form field of not less than 45 degrees in all meridians from the point of fixation.
   3 The presence of binocular single vision.
   4 Ability to distinguish red, green and yellow.
   5 Night blindness not to be present.
   6 Glasses when required be worn while driving and those employed in public transportation be provided with an extra pair.

B. Visual Standards for Limited License:
   1 Visual acuity of not less than 20/65 Sn. in the better eye.
   2 Field vision of not less than 60 degrees horizontally and 50 degrees vertically from point of fixation in one eye.
   3 Diplopia not to be present.
   4 Glasses to be worn when prescribbed.
   5 Coordination of eye, mind and muscle to be fully adequate to meet the practical visual road tests.
   6 A limited license not to be issued to those employed in public transportation.

C. Renewals, Retesting and Reexaminations:
   1 Renewals of license to be issued at least every third year. The applicant shall with each renewal make a declaration that he knows of no visual defect which has developed during the past year.
   2 Retesting of acuity to be made at least every six years.
   3 If any visual defects have developed, an examination by an ophthalmologist and the report thereof, to be required before reissuing the license.
   4 License to state thereon the specific limitation for driving.

—*Jour. A. M. A.*

COMMUNICATIONS

Sir:

I am enclosing a copy of the telegram received by me from Dr. Olin West of Chicago. Please give this space in the Southwestern Journal so that all the members of the Society will be sure to get this information.

Thanking you in advance, I remain,

Fraternally yours,

NEW MEXICO MEDICAL SOCIETY.
L. B. Cohenour, M. D.
Secretary-Treasurer.

CHICAGO, ILL.

NEW MEXICO MEDICAL SOCIETY DR. L. B. COHENOUR 219 WEST CENTRAL AVE ALBUQUERQUE N MEX

THE SPEAKER OF THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION HAS CALLED A SPECIAL SESSION OF THE HOUSE OF DELEGATES TO BE HELD IN CHICAGO SEPTEMBER 16 1938 STOP COPY OF THE OFFICIAL CALL IS BEING FORWARDED STOP PLEASE ADVISE OTHER OFFICERS AND MEMBERS OF OFFICIAL BODIES OF YOUR ASSOCIATION AS WELL AS GENERAL MEMBERSHIP SO FAR AS POSSIBLE

OLIN WEST—SECRETARY
AMERICAN MEDICAL ASSOCIATION

NEWS

General

The Twenty-third International Assembly of the Inter-State Postgraduate Medical Association of North America will be held in the public auditorium of Philadelphia, Pennsylvania, October 31, November 1, 2, 3 and 4, 1938. All scientific and clinical sessions will take place in the auditorium. Hotel headquarters will be the Benjamin Franklin Hotel.

The members of the medical profession of Philadelphia are correlating for the clinics, an abundance of hospital material representing various types of pathological conditions which will be discussed by the contributors to the program.

In the neighborhood of eighty distinguished teachers and clinicians will appear on the program, a tentative list of which may be found in the advertising section of this Journal. The subjects and speakers have been selected to consider practically all the subjects of greatest interest to the medical profession in general.

A full program of scientific and clinical sessions will take place every day and evening of the Assembly starting each morning at 8:00 o'clock. On account of the fullness of the program, restaurant service will be available at the auditorium at moderate prices.

The members of the profession are urged to bring their ladies with them as a very excellent program is being arranged for their benefit by the ladies' committee. Philadelphia has many places of historic and other interests, which will make this year's program especially attractive to them.

Pre-assembbly and post-assembly clinics will be held in the Philadelphia Hospitals on Saturday, October 29 and Saturday, November 5.

It is important that hotel reservations be made early by writing Mr. Thomas E. Willis, Chairman of the Hotel Committee, Chamber of Commerce Building, 12th and Walnut Streets, Philadelphia, Pa.

The Association, through its officers and members of the program committee, extend a very

hearty invitation to all members of the profession in good standing in their State and Provincial Societies to attend the Assembly. The registration fee is $5.00.

BOOK NOTES

TUBERCULOSIS AMONG CHILDREN AND YOUNG ADULTS. By J. Arthur Myers, Ph.D., M.D., F.A.C.P., Chief of Medical Staff and Director of Tuberculosis Activities, Lymanhurst Health Center, Professor of Preventive Medicine, University of Minnesota. Cloth, price $4.50. Pp. 401, many illustrations. Charles C. Thomas, Baltimore, Maryland.

The first edition of this excellent book by Dr. Myers was published in 1930. It was so well received that the edition was soon exhausted. This, the second edition, has been largely re-written, and the text enlarged to include a more exhaustve study of tuberculosis in young adults. We know of no man in the medical profession who is so well qualified to write on these subjects, as is the author of this book. He has spent the greater part of his professional life in study and research in this particular phase of tuberculosis.

His remarkable work carried out in the Lymanhurst Health Center, in Minneapolis, is outstanding and has resulted in a wealth of information which Dr. Myers has so graciously passed on to the profession in this book.

The first chapter on "Recent Progress in Tuberculosis and its Practical Application in Diagnosis, Treatment and Prevention," and the second chapter on "Sensitization and Immunity," are intensely interesting and most valuable. In fact, the entire text brings information which should be helpful to every physician, no matter in what special line he is interested.

It is quite evident, that if tuberculosis is to be controlled, we must start with infancy and childhood; therefore, the information contained in this book is almost indispensble to the phthisiologist, the pediatrician and the public health worker.

—*R. B. H.*

DISEASES OF THE SKIN FOR PRACTITIONERS AND STUDENTS. By George Clinton Andrews, A.B., M.D., Associate Professor of Dermatology, College of Physicians and Surgeons, Columbia University; Chief of Clinic, Department of Dermatology, Vanderbilt Clinic; Fellow of the American Medical Association, of the American College of Physicians, and of the New York Academy of Medicine. Second edition, entirely reset. 899 pages with 938 illustrations. Philadelphia and London: W. B. Saunders Company, 1938. Cloth, $10.00 net.

Andrew's first edition has for several years been accepted as a standard text and reference book on dermatology and syphilology. This new second edition offers many new advances in this field and in my opinion is much better suited to both the general practitioner and to those who are engaged in the practice of the specialty exclusively.

The new edition is smaller, yet it contains descriptions of some seventy-five new diseases not described in the first edition. New chapters on virus diseases, vitamin deficiencies, and cutaneous infiltrations with products of metabolism, have been added. Several of the chapters on the commoner dermatoses have been entirely re-written with many of the older ileas being supplanted by newer ones concerning diagnosis, etiology and treatment.

The photographs in the new edition are much superior to those encountered in most books on dermatology, and are of considerable aid in fixing characteristics of the dermatoses in the reader's mind. I cannot praise these photographs too highly.

Slight change in the arrangement of the reading matter simplifies the studying of this book. The histo-pathology of each disease is now discussed fully under the disease instead of being listed under a general chapter on pathology at the beginning of the book. In most instances histologic drawings or photo-micrographs are used to illustrate the microscopic appearance.

Information concerning roentgen-ray and radium therapy is much more concise and to the point. In the first edition much space was allotted to the physics and technicalities of x-ray and radium, things that are of interest chiefly to the physicist rather than to the practitioners of medicine. In the second edition more attention is given to the indications and contra-indications, technique of treatment and dosage, which makes it of more interest and value to us as practitioners. The section on ultraviolet therapy and surgical diathermy have likewise been condensed.

The author's discussion of the subject of syphilis is very thorough and yet concise and to the point. The importance and commoner cutaneous manifestations are discussed adequately in all the different stages, accompanied by multiple illustrative photographs to impress upon us the idea in the author's mind. Many new and useful ideas are brought out concerning both diagnosis and treatment. In this era of campaigning against the number one social disease in which all physicians are called upon by the laity concerning certain pertinent points and facts, it is certainly of distinct benefit to be able to review the newer ideas and facts without having to wade through works that are too voluminous. This section should be of special interest to the general practitioner.

—*R. P. H.*

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. A complete dictionary of the terms used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, Nursing, Veterinary Science, Biology, Medical Biography, etc. By W. A. Newman Dorland, A.M., M.D., F.A.C.S., Lieut.-Colonel, M.R.C., U. S. Army; Member of the Committee on Nomenclature and Classification of Diseases of the American Medical Association; Editor of the "American Pocket Medical Dictionary". With the collaboration of E. C. L. Miller, M.D., Medical College of Virginia. Eighteenth edition, revised and enlarged. 1607 pages with 942 illustrations, including 283 portraits. Flexible and stiff binding. Philadelphia and London: W. B. Saunders Company, 1938. Plain, $7.00 net. Thumb indexed, $7.50 net.

This is the eighteenth edition of the most valuable book any practitioner of medicine can possess. There are more than 5,000 new words listed in this text. Pronunciation of words is featured throughout. The terminology conforms to the standard of various scientific bodies who have adopted such standards as part of their functions. Various tables of handy reference value are found throughout the book. Especially interesting are the short bio-

graphical sketches of well-known men in medicine. 283 portraits of these physicians are printed. If a reader expects to thoroughly understand modern medical literature, it would be incumbent on him to possess and use this latest compilation of medical terms. —*S.*

A TEXTBOOK OF BACTERIOLOGY. By Thurman B. Rice, A.M., M.D., Professor of Bacteriology and Public Health at the Indiana University School of Medicine. Second edition, revised. 563 pages with 121 illustrations. Philadelphia and London: W. B. Saunders Company, 1938. Cloth, $5.00 net.

This volume of five hundred and sixty-three pages is well designed to accomplish the author's purpose. Namely, to give the student a comprehensive survey of pathogenic bacteriology without too much detail. The various classes of bacteria are well described. Methods of identifying them are clear. Treatment is concisely outlined. The theories of immunity are covered and their practical application in treatment and diagnosis is set forth. Each subject is considered in the light of fundamentals that the practicing physician should be familiar with. No attempt has been made to make the column a reference book for the bacteriologists.

The volume fills its place as a text for students and can be well recommended for the physician who wishes to have a not too technical reference or to review the subject of bacteriology.

—*L. O. D.*

OUTLINE OF ROENTGEN DIAGNOSIS, AN ORIENTATION IN THE BASIC PRINCIPLES OF DIAGNOSIS BY THE ROENTGEN METHOD. By Leo. G. Rigler, B.S., M.B., M.D., Professor of Radiology, University of Minnesota. Minneapolis, Minnesota.

The book is well written and comprehensive with simple, direct phraseology.

While it is primarily a student's edition, it would be a valuable asset to the doctor who specializes in radiology and the general practitioner.

The normal and pathological findings are briefly but clearly defined and the indications and contra-indications for taking x-rays are well outlined.

A paragraph on head injuries should well be brought to the attention of every practicing physician. "Aside from medico-legal considerations, the importance of demonstrating any fracture of the skull other than the depressed type is very questionable. The treatment is dependent entirely upon the patient's clinical condition. It is inadvisable, therefore, to subject a patient suspected of having a skull fracture to any considerable manipulation immediately after the injury, merely for the purpose of roentgen examination. When all danger is past, a complete examination may be made."

The above is characteristic of the conservatism of the author. —*C. H. M.*

PATHOLOGICAL TECHNIQUE. By Frank Burr Mallory, A.M., M.D., S.D., Consulting Pathologist to the Boston City Hospital, Boston, Mass. 434 pages with 14 illustrations. Philadelphia and London: W. B. Saunders Company, 1938. Cloth, $4.50 net.

This volume of four hundred and thirty-four pages proves to be one of the most helpful of the recent volumes on pathological technique. The various steps in the preparation of microscopic sections both for routine use and for special demonstration of the various elements are clear cut and concise. Little is left to the imagination, so that careful following of the directions should result in excellent products. The minutae of various procedures has not been overlooked. Many insignificant "tricks" of the technician are described, all of which make for better technique. A paritcular interest to the technician is the mechanism of action of the various staining procedures, and the book is found to be definitely more than just a summary of technical steps. Of particular interest is the detailed discussion of post-mortem technique which covers about seventy pages. Likewise, there is an excellent discussion of the major points in photography of pathological specimens. The excellent features of the book may be summarized by noting that it is complete, pay considerable attention to detail that is often lacking in many descriptions of technical methods, is excellent in make-up and yet is not verbose. The main criticism is that it is sometimes difficult to locate just the points sought; although admittedly the wealth of techniques that are described makes it difficult to adequately classify and arrange them. —*L. O. D.*

"IS THIS PRODUCT COUNCIL-ACCEPTED?"

This is the first question many physicians ask the detail man, when a new product is presented.

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Mead Johnson & Company cooperates with both Councils, not because we have to, but because we want to. Our detail men can always answer you, "Yes, this Mead Product *is* Council-Accepted."

An emergency operating room will be part of the completely equipped hospital on Treasure Island in San Francisco Bay throughout the 288 days of the World's Fair of the West.

A staff of several nurses, internes and physicians will be in attendance at the Treasure Island hospital throughout the 1939 World's Fair of the West which starts next Febrlary 18.

Control of Syphilis!

A most important factor in the diagnosis and control of syphilis is reliable blood, darkfield, and spinal fluid examinations.

We run daily Wassermann (Kolmer modification) and Kahn tests.

Results are sent the referring physician by wire (at our expense) when requested to save time.

A supply of Keidel tubes for blood withdrawal, in mailing containers, sent cheerfully on request.

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EL PASO, TEXAS

GEORGE TURNER, M. D. DELPHIN von BRIESEN, M. D.

PACIFIC MUTUAL NEWS

By Frank J. Schwentker

When a doctor tells me he does not want more life insurance I quite agree with him. I feel he is sincere in his statement, for I don't want more insurance myself. BUT both he and I want the things life insurance will do for us.

We don't wont more life insurance but if we are taken out of the picture prematurely, we want our wife to have a guaranteed income that will enable her to complete the rearing of our children and maintain her in comfort as long as she lives.

We don't want life insurance but we want to guarantee a college education for our children so they will have a fair break in this highly competitive world. We don't want life insurance, but when we reach age 60 or 65 and retire from business, we want a dependable and guaranteed income which will continue as long as we live.

No, we don't want life insurance as such, but we do want all the things life insurance will do for us. And life insurance is the only type of property which matures according to need, rather than date, and will deliver guaranteed income to replace our earned income whenever it is terminated by accident, sickness, old age, or premature death.

Our underwriters are trained not to sell life insurance as such, but to arrange a financial program which will accomplish the hopes and dreams and ambitions you have for yourself and your family.

The F. B. Schwentker Agency,
711 Title & Trust Bldg.,
Phoenix, Arizona.

The Schwentker-Bruce Agency,
915 Mills Building,
El Paso, Texas.

SCRAPS

THE SILLY SEASON

The Boston Boys, in their erudite New England Journal of Medicine, get hot under the collar over this business of Carrying Coals to Newcastle—

Hay as a merchantable commodity may have been relegated mainly to the rural regions, but haywire remains with us as the rather pathetic mainspring of the silly summer season. To what extent haywiriness is a natural and inevitable development of the dog days—a damp humor born of heat and humidity—is difficult to say. Undoubtedly weather and the slack season are behind this trend toward the twisted shackles of dessicated timothy, redtop and alfalfa, but there are other motivations.

We suspect that business might pursue its sane if somewhat humdrum way, were the perspiring penpushers of a bogged-down Fourth Estate not afflicted with the scabietic urge to make something out of nothing. It is interesting, but rather pitiful to note what putty we are in the hands of the penny-thriller purveyors. A preadolescent moving picture actress develops the sniffles in Boston; a physician must be rushed from Hollywood by plane. No reflection on the Boston doctors, you understand, but in the cinema business, if it doesn't cost money, it's no good, and a headline a day keeps the doldrums away. . . . . .

Even with no personal experience to guide us in the more remote zones of antiquity, we believe that the sweaty populace was always ready to clap its chopped hands and throw its hats into the air. Sensationalism has the geratest appeal, and the press of the country is making no high effort to direct the thoughts of the nation into inspiring or productive channels. The average citizen wants his news to be of about the same consistence as the pulp on which it is printed, with his taste perhaps at its lowest ebb in the beach-pajama season.

LOVE'S SEAT

Wit and humor of the Great Southwest, as delicately expressed by a brother physician discussing "Anal Infections" before the New Mexico state meeting in June:

It used to be thought that our emotional states had their origin in the heart. In the Spring when a young man's fancy lightly turns to thoughts of love, he does it with all his heart. One man has a kind heart, another one a Big Heart, or a Warm Heart, or a Tender Heart! while someone else is hard-hearted or has a heart of stone, all expressions testifying to man's localization of his emotional states. Then our Laboratory brethren came along and took all of the romance and hatred out of a man's heart and put them in his endocrine glands. Romeo in the Balcony scene placed one hand over his heart as he extended the other to Juliet standing above. With this endocrine stuff he had no place to put it. Now I know that gloom and irracibility are definitely located along the pectineal line and love may be localized there also. Hitler's

great purge in Germany some years ago would suggest it. And if it is so, then Romeo will again have a place to put his hand.

RURAL REMEDIES

The learned Science of Materia Medica gets valuable additions, as noted by our Phoenix sleuth, Dr. H. Y.—

A rifle shot wound in the leg of an Indian near Laguna, New Mexico, was satisfactorily treated with poultices made out of community feces. . . I saw the case. . . Believe it or not.

* * *

A grand old doctor who has "practiced medicine" 60 years told me long ago that about the only drugs that had not lost their virtue through the years were castor oil, Lydia Pinkhams Compound, Rocky Mountain Tea, turpentine and honey.

* * *

My mother rubbed equal parts of possum oil and turpentine on my throat for croup many a time. . . It usually cured me, but sometimes it didn't so then she unscrewed the coal oil lamp and had me drink a teaspoonful of the oil—*that* always cured me. . . And as I grew older and kept on having croup, I conveniently learned to never go to sleep at night without having the lamp in reach of my bed.

* * *

"If you'll put a good grade of camphor gum over your belly button, it'll cure your hayfever." It must be worn constantly "right on the nabel". . . A full blood Nazi girl told me this in my office yesterday. . . She said she knew a man who wore his camphor for two months and then had to take a bath, and he forgot to put his camphor back on again and he sneezed for nearly twenty-four hours. . . She said that when the camphor touched his nabel again he stopped about half way in one of his sneezes. . . "It works", she said.

Expert medical care and modern hospitalization will be available to all visitors at the 1939 Golden Gate International Exposition on San Francisco Bay, at a twelve-bed hospital on Treasure Island.

Physicians, of all men, are most happy: what good success soever they have the world proclaimeth, and what faults they commit the earth covereth.—Paxon Quarrel.

INTERNATIONAL MEDICAL ASSEMBLY

Inter-state Postgraduate Medical Association of North America

Public Auditorium, Philadelphia, Pa. Oct. 31, Nov. 1-2-3-4, 1938.

Pre-Assembly Clinics, October 29; Post-Assembly Clinics, November 5, Philadelphia Hospitals

President, Dr. Elliott P. Joslin; President-Elect, Dr. George W. Crile
Chairman Program Committee, Dr. George W. Crile; Managing-Director, Dr. William B. Peck
Secretary, Dr. Tom B. Throckmorton; Director of Exhibits, Dr. Arthur G. Sullivan
Treasurer and Director of Foundation Fund, Dr. Henry G. Langworthy
Chairman Philadelphia Committees, Dr. Louis H. Clerf

ALL MEDICAL MEN AND WOMEN IN GOOD STANDING CORDIALLY INVITED

Intensive Clinical and Didactic Program by World Authorities

The following is a major list of members of the profession who will take part on the program:

Alfred W. Adson, Rochester, Minn.
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George Blumer, New Haven, Conn.
Peter T. Bohan, Kansas City, Mo.
William F. Braasch, Rochester, Minn.
Richard B. Cattell, Boston, Mass.
Henry A. Christian, Boston, Mass.
Arthur C. Christie, Washington, D. C.
Edward D. Churchill, Boston, Mass.
Louis H. Clerf, Philadelphia, Pa.
William Cone, Montreal, Canada.
John S. Coulter, Chicago, Ill.
George W. Crile, Cleveland, Ohio.
Elliott C. Cutler, Boston, Mass.
Walter E. Dandy, Baltimore, Md.
William Darrach, New York, N. Y.
Vernon C. David, Chicago, Ill.
Loyal Davis, Chicago, Ill.
Robert S. Dinsmore, Cleveland, Ohio.
Claude F. Dixon, Rochester, Minn.
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Eldridge L. Eliason, Philadelphia, Pa.
William H. Erb, Philadelphia, Pa.
John F. Erdmann, New York, N. Y.
A. Carlton Ernstene, Cleveland, Ohio.
Clarence B. Farrar, Toronto, Canada.
John C. Gittings, Philadelphia, Pa.
Russell L. Haden, Cleveland, Ohio.
William D. Haggard, Nashville, Tenn.
Samuel F. Haines, Rochester, Minn.
George A. Harrop, New York, N. Y.
Charles G. Heyd, New York, N. Y.
Fred J. Hodges, Ann Arbor, Mich.
Chevalier Jackson, Philadelphia, Pa.
Chevalier L. Jackson, Philadelphia, Pa.
Elliott P. Joslin, Boston, Mass.
Frederick J. Kalteyer, Philadelphia, Pa.
Floyd E. Keene, Philadelphia, Pa.
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Frank H. Lahey, Boston, Mass.
Dean Lewis, Baltimore, Md.
Walter I. Lillie, Philadelphia, Pa.
Perrin H. Long, Baltimore, Md.
Warfield T. Longcope, Baltimore, Md.
William E. Lower, Cleveland, Ohio.
Charles W. Mayo, Rochester, Minn.
Irvine McQuarrie, Minneapolis, Minn.
James H. Means, Boston, Mass.
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John J. Moorhead, New York, N. Y.
George P. Muller, Philadelphia, Pa.
Clay Ray Murray, New York, N. Y.
John H. Musser, New Orleans, La.
Howard C. Naffziger, San Francisco, Cal.
Frank R. Ober, Boston, Mass.
Eric Oldberg, Chicago, Ill.
Oliver S. Ormsby, Chicago, Ill.
Hubley R. Owen, Philadelphia, Pa.
Wilder Penfield, Montreal, Canada.
George E. Pfahler, Philadelphia, Pa.
Fred W. Rankin, Lexington, Ky.
Robert F. Ridpath, Philadelphia, Pa.
David Riesman, Philadelphia, Pa.
Leonard G. Rowntree, Philadelphia, Pa.
Thomas H. Russell, New York, N. Y.
E. Kost Shelton, Los Angeles, Cal.
Fred M. Smith, Iowa City, Iowa.
Marius N. Smith-Petersen, Boston, Mass.
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